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Original Investigations on the Natural History, (Symptoms and Pathology) of Yellow Fever. 1854-1894.

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ORIGINAL INVESTIGATIONS ON THE NATURAL HISTORY, (SYMPTOMS AND PATHOLOGY) OF YELLOW FEVER. 1854–1894.

Yellow fever (vomito; vomito prieto; vomito negro, fièvre matelotte; febris flava; fièvre jaunne; gelbes fieber; febbre grilla; febris icterodes; typhus icterodes; typhus tropicus; typhus d' Amérique; fiebre amarilla; coap de banc; febris maligna biliosa Americana flava; bulam fever; hemogastric pestilence; pestilentia hemogastrica; synochus icterodes; causus; febris flava Americanorum, epanetus malignus flavus; typhus jaune; ichwaites erbrechen westendisches fieber; fiebre yialla; black vomit; malignant yellow fever; pestilential fever; epidemic yellow fever; specific yellow fever.)

DEFINITION.

A pestilential fever, of a continuous and specific type, originally developed in tropical and insular America; confined to definite geographical limits, and dependent in its origin and spread upon definite degrees of temperature, and capable of transportation and propagation on ships and in towns and cities in those portions of North and South America which lie between 45° N. latitude and 35° S. latitude. The disease has been limited chiefly to the coast of tropical America, rather from the number and position of the commercial centers, than from any climatic causes adverse to its propagation elsewhere. It has been imported chiefly from the Antilles, and from the cities of tropical America by ships into the Gulf and Atlantic cities of the United States, and into Cadiz, Carthagena, Barcelona, Gibraltar, Lisbon, Saint Nazain in the department of the lower Loire, and into Plymouth and Southampton in Europe; and it has been imported and become epidemic as far south as Montevideo and Buenos Ayres in South America.

As a general rule, it has originated and become endemic in cities situated in low, unhealthy malarious districts on the sea or river coasts of insular and tropical America, and has rarely occurred at an elevation of 2,500 feet above the level of the sea; in Jamaica it has prevailed at Stoney Hill, 1,300 feet above the sea, and at Newcastle 4,000 feet above the sea; according to Alexander Humboldt, it has never ascended in Mexico to 3,044 feet above the level of the sea, below which limit the Mexican oaks do not flourish, showing that the constant average temperature below this is of a tropical character.

In the United States the disease has never in its epidemic form reached an elevation of 500 feet, and it has been observed that its epidemic course or limit coincides to a certain extent with the range of the growth of the live-oak, the cypress and the long moss. The inhabitants of the barren rock of Gibraltar, at an elevation of between 1,400 and 1,439 feet above the level of the sea, have been desolated by this disease upon more than one occasion. It is certain that yellow fever has prevailed in the celebrated table land of Caracas, 3,000 feet above the level of the sea. In the remarkable epidemic of yellow fever which prevailed in Peru in 1855 and 1856, the disease passed over the barrier of the Andes, committing fearful ravages in Andine and trans-Andine regions, at elevations of 14,000 feet above the level of the sea. The stereotyped expressions of systematic writers, as to the limitation of yellow fever to certain elevations and to the sea and river coasts must in the light of the preceding facts be abandoned.

Yellow fever presents two well defined stages; the first characterized by severe pains in the head, confined chiefly to the orbits and forehead, back and lower extremities, a peculiar shining or drunken appearance in the eyes, rapid circulation, elevated temperature, and increase of those constituents as urea, uric acid, phosphoric acid and sulphuric acid which result from the increased chemic changes induced by the febrile poisons; and which stage may extend from 36 to 150 hours, without any distinct remissions, according to the severity of the disease; the second characterized by depression of the nervous and muscular forces, and of the general and capillary circulation, capillary congestion, slow and intermittent pulse, jaundice, a purplish and yellowish mottled appearance of the surface, urinary suppression, albuminous urine loaded with granular casts of the urinary tubes, fatty degeneration of the heart, liver and kidneys, defibrination of the blood, passive hemorrhages from ears, stomach and bowels, nares, tongue, gums, uterus, vagina, gall bladder and anus, and in extreme cases from the eyes, ears and skin, black vomit, interstitial hemorrhages, delirium, convulsions and coma. In its origin and propagation, it appears not to be dependent on those conditions and causes, which generate malarial paroxysmal fever, from which it differs essentially in symptoms and pathology.

One of the prominent symptoms of the first stage is that rapid increase of the pulse, within the first few hours of the febrile excitement, and the progressive diminution of the beats of the heart, even while the temperature progressively rises; and in like manner, the slow and feeble action of the heart constitutes a prominent and striking symptom of the second stage. Yellow fever, in common with such contagious diseases as smallpox, measles and scarlet fever occurs as a general rule, but once during life, and may be propagated by contagion; it differs, however, from the exanthematous diseases, in that it has never been known to propagate beyond 48° N., and 38° S. latitude, nor below a temperature of 65 degrees F. The symptoms of yellow fever may be divided for description and investigation into:

1. Those manifested during the period of incubation, or that period of time which elapses between the introduction of the poison inducing the disease and the actual manifestation of the febrile phenom-

ena. As this stage is of uncertain duration, the disease sparing neither age nor sex, those only being exempt from its influence who have, at some former time, been subjected to the action of the yellow fever poison, and its attacks being confined neither to the night or the day, nor to any special state of the system, whether of fasting or repletion, of plethora or anemia, of robust health or chronic disease; and as the disease often attacks suddenly those who are apparently in perfect health, who are seized with intense headache, chill, shivering pain in the limbs and back, followed by rapid elevation of temperature, increased action of the circulation, animated congested countenance, red, glistening suffused eyes, intense thirst, anorexia, uneasiness of the epigastrium, nausea and vomiting; the investigation of the phenomena of the stage of incubation is attended with great difficulty and much uncertainty.

While it must be admitted, that during the time which elapses from the moment of the effective action of the cause, to that of the manifestation of the disease, changes are produced in the blood and nervous system and which, although producing no recognized obstacle to the healthy performance of the functions, are not the less real, and increase up to the moment of the final explosion of the disease; but as to the nature of these changes in the system, we know nothing, and the subject is open for investigation. The difficulty of such an inquiry is clearly recognized, when we consider the want of knowledge of the nature of malaria, of the constituents of emanations from the soil, and of other imputed sources of pestilential diseases; the mist of ignorance and prejudice involving these agents, and exaggerating their influence, and their inferred nature and operation, without further proof of their existence than certain effects which have been imputed to them either upon insufficient data, or even without the smallest evidence.

A sure foundation for the investigation will be reached, when the yellow fever poison is isolated; either from the soil, waters, or atmosphere, or from the diseased human body. Our knowledge has not and will not be advanced by hypothetical discussions as to the mineral, vegetable or animal origin and nature of the yellow fever poison; and the time wasted in such arguments would be much better employed by careful physical, chemic, microscopic and physiologic investigation of the waters, atmosphere and soil and of the minute flora and fauna of those regions and places in which the disease is endemic and epidemic.

2. The symptoms manifested during the exhibition of the first disturbances in the nervous and circulatory systems are characterized by chilly sensations, and changes of temperature, and severe pains in the head and loins, restlessness, mental depression and anorexia. The symptoms of this stage, which may be called that of chill or primary depression, are not uniform; in some cases, and even in those of the greatest violence, chill and chilly sensations are entirely absent; in others, the chill amounts only to a sensation of coldness, sometimes slight; at others, deep, penetrating and stupefying, or alternating with flushes of heat and crawling sensations; while in others, the febrile excitement is preceded by chills of greater or less severity and duration, with a constriction or contraction of the extreme peripheral vessels, and a shriveled appearance of the skin; in some cases again, the disease is ushered in by giddiness, bearing some analogy to that produced by inebriating liquors; and in others it has been known to have commenced with convulsions.

The presence or absence of chills and chilly sensations can not, therefore, be regarded as characteristic of yellow fever; and the chills, when most strongly marked, appear to differ materially from the chill of paroxysmal malarial fever, in that there is an absence of uniformity in the phenomena, and that the chill or cold stage of yellow fever partakes rather of the aberrated nervous actions and sensations, referable to the action of the causes producing the intense pain in the head and loins, than that marked depression of the temperature of the periphery and elevation of the temperature of the internal organs, even to the degree of 107 degrees F., which I have shown by careful observation to be characteristic of the[1] true malarial paroxysmal chill.

The chill and chilly sensations of the early stages of yellow fever, appear rather to be related to the same manifestations of aberrated nervous action in the early stages of various acute diseases, as pneumonia, pleuritis and smallpox. As these symptoms are often absent in yellow fever, as they occur in the earlier stages, and as they are in most cases of short duration, it is not often that the physician has the opportunity of investigating the phenomena by means of the thermometer and sphygmograph, and by the careful collection and analysis of the urine during this period.

3. The symptoms of the stage of reaction, or active febrile excitement following the chill and chilly sensations, or arising as it were spontaneously, with but few premonitory symptoms beyond uneasiness and pain in the head, loins, and limbs, and general prostration and languor, characterized by rapid elevation of temperature, which is maintained with slight morning and evening oscillations, and with a progressive descent to the normal standard, for periods varying in different cases, from one to six days. During this stage of active fébrile excitement, profound changes are induced by the yellow fever poison, by the products to which it gives rise in the blood and organs. The maximum elevation of temperature is rapidly attained, and may range during the first days of the disease, according to its severity, from 102 degrees F. to 110 degrees F. in the axilla, and it is prob-

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[1] Observations on some of the Physical, Chemical, Physiological and Pathological Phenomena of Malarial Fever, Transactions AMERICAN MEDICAL ASSOCIATION, 1859.

able that the internal organs attain a still higher temperature. As a general rule, from the third to the fifth day, the temperature steadily falls and sinks to the normal standard and even below; in some fatal cases it rises again toward the end, rarely, however, reaching or exceeding 104 degrees F., and never attaining the high degree of temperature characteristic of the stage of active febrile excitement.

The supervention of an inflammatory disease or the occurrence of an abscess will, in like manner, cause a progressive elevation of temperature, with slight evening exacerbations; but as a general rule, the secondary elevation of temperature thus caused never attains the maximum of the stage of active febrile excitement.

When malarial paroxysmal fever is engrafted on yellow fever in its later stages, or during the period of convalescence, the temperature during this hot stage may even exceed the maximum of the hot stage of yellow fever, but will be characterized by sudden periodic depressions and elevations, recurring at definite intervals. The pulse, at the commencement of the attack, when the period of reaction or active chemic change has fairly set in, is often rapid (ranging from 100 to 140 per minute), strong, tense and full; in other cases it is rapid but feeble and compressible; the increase in the frequency of the pulse does not, however, as a general rule, continue to correspond with the elevation and oscillations of temperature; and in many cases, the remarkable phenomenon is witnessed of the pulse progressively decreasing in frequency, and even descending below the normal standard, while the temperature is maintained at an elevated degree; and in the examination and comparison of these discrepancies between the frequency and character of the pulse, and the oscillations of the temperature the observer is forced to the conclusion that during the hot stage, some poison or depressing agent is acting upon the heart, or upon the nerves which supply its muscular structures and regulate its actions, thus deranging the nutrition and molecular structure of the muscular fibers. On the other hand, the pulse frequently increases in rapidity, but diminishes in force near the fatal issue.

The occurrence of copious hemorrhages from the stomach and bowels may be attended with sudden depressions of temperature and increase in frequency, but diminution in force and fullness of the pulse.

The stage of febrile excitement is not of fixed duration, and may, in some cases of the gravest character, appear to be comparatively mild and unimportant, attracting so little attention, and the pains, wherever seated, may be so mild, and there may be such an absence of agitation and delirium, and the strength of the patient may be diminished to so slight a degree, that both patient and physician may be deceived and the patient may die without taking to his bed, *on foot*, as it were.

Case 1.—Louis[2] recounts the case of Dr. Mathias, who died after an illness of four or five days, and who experienced no other symptoms but some pains in the calves of the legs and a suppression of urine. He had no nausea; he did not vomit. His mind was clear through the whole course of the disease. He noticed the continuance of the suppression of urine; dictated three or four letters to a friend; begged him to write rapidly, so that he might sign his name, then devoted a little time to an affectionate intercourse with this friend, and soon after, unable to speak, he thanked his friend with a sign, and in a quarter of an hour he was dead.

Case 2.—In the month of October, 1870, a stout Irishman walked up from the gate of the Charity Hospital of New Orleans, and entered one of the wards on the third floor up stairs, presenting to the casual observer no special marks of disease beyond some congestion of the capillaries of the face and extremities, a dusky, livid hue, a slight increase of heat, and restlessness. A few hours after entering this ward, he vomited about one quart of black vomit, and died in convulsions twenty-four hours after his entrance into the ward. The black vomit ejected during life, as well as the portion which filled the stomach after death, presented the appearance and character of dark grumous blood, was of high specific gravity and alkalin reaction, contained both ammonia and urea, and also an immense number of altered, colored blood corpuscles. Careful microscopic examination with lenses of different powers, ranging from one-quarter to one-eighteenth of an inch, revealed no animalculæ or vegetable organisms (vibrios and fungi are frequently present in black vomit, but as far as my observations extend, they have nothing to do with its causation). The superior portions of the body of this patient, after death, presented a deep golden yellow color, while the dependent portions presented a purplish, mottled, ecchymosed and congested appearance; the heart was pale, yellow and flabby, and contained many oil globules, and the muscular structures or fibers presented an altered appearance under the microscope; the transverse striæ being less distinct, and the interior of the muscular fibrillæ, as well as the connective tissues, being loaded with oil globules. The kidneys were pale and of a yellow color, resembling that of the heart, and the excretory tubes were filled with albuminous and fibroid granular matter, excretory cells, and oil globules. The bladder contained no urine, there having been complete urinary suppression since the entrance of the patient into the hospital. The liver presented the usual yellow color and bloodless appearance of this organ in yellow fever, and the liver cells contained an abnormal amount of oil.

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[2] Documens Recuellis par MM. Chervin, Louis et Trousseau, Membres de la Commission Médicale Francaise envoyée á Gibraltar pour observe l'epidemie de 1828, et par M. le Dr. Barry. Two Vols. Paris, 1830. Researches on the Yellow Fever of Gibraltar, by Dr. Ch. A. Jones.

In these changes yellow fever resembles acute phosphorus poisoning.

The gall-bladder, as is usual in this disease, contained only a small quantity of yellow bile, not exceeding one hundred grains in amount; while in malarial fever, on the other hand, the gall-bladder, after death, is almost universally distended with dark, greenish-black bile of high specific gravity, and generally about one thousand grains in amount.

In some cases of yellow fever which I have observed in New Orleans, the symptoms have rapidly changed from active excitement to utter prostration, and the patients have been speedily destroyed by hemorrhage from the bowels, without any vomiting of the altered blood and gastric secretions which constitute black vomit. I have also observed cases, in which hemorrhage of black blood had taken place in the gall-bladder. In such cases the gall-bladder has been distended with a dark grumous liquid, which upon microscopic and chemic examination proved to be defibrinated blood, without any admixture of bile.

As far as my observations extend, *suppression* of urine is a universally fatal symptom, and is the prime cause, not only of the copious hemorrhages from the stomach and bowels, but also of various alterations of the blood and nervous disturbances, and of the sudden and apparently inexplicable deaths.

Suppression of urine in yellow fever is attended with the accumulation of the constituents usually eliminated by the kidneys, as urea in the blood, brain, liver and heart and muscular organs, as I have demonstrated by numerous analyses. Bile also accumulates in such cases. The presence of the coloring matter of the bile in large amounts in the circulatory fluid may be demonstrated by a simple experiment; thus, if a drop of blood be dropped upon a piece of filtering paper, a brilliant red central spot will mark the presence of the colored blood corpuscles, and this will be surrounded by a bright golden border or ring, due to the absorption of the serum deeply tinged with bile.

The peculiar indifference and even intoxication which mark the cases of suppression of urine are referable chiefly to the retention of the constituents of the urine and the bile.

The changes of the blood in such cases of yellow fever are characterized chiefly by great diminution of the fibrin, which, in some cases, I have found to be not one hundredth of the usual amount; and by the abnormal amounts of urea and ammonia and of the sulphates, phosphates and extractive matters.

I have found the stomach and intestines so charged with ammonia, even when examined within three hours after death, that a rod dipped in hydrochloric acid, held over the mucous membrane of the stomach and intestines, gave forth dense white fumes (chlorid of ammonium).

It is without doubt, true, that the poison of yellow fever acts as a direct irritant or poisonous agent upon the mucous membrane of the stomach, not only by deranging its nervous supply and capillary circulation, but also by directly altering the nutrition and secretion of its active cellular elements; but at the same time it is also true that the poison acts equally, if not to a more marked degree, upon the liver and kidneys, thus causing in many cases complete suppression of the function of both organs, and the consequent contamination of the blood with bile, urea and extractive matters and ammonia.

The excrementitious matters thus retained in the blood not only exert a depressing action upon the nervous and circulatory systems, and change the important nutritive actions of the colored blood corpuscles, but the urea, eliminated as such, and as carbonate of ammonia by the gastro-intestinal mucous membrane, alters the constitution of the intestinal and gastric juices, and directly the mucous membrane of the stomach and bowels.

Analogous cases to those just described are witnessed in the action of certain violent poisons, which act alike upon the nervous system and the blood.

It is but just for us to view such cases as a febrile action of an infectious character, deriving its peculiar characters and malignity, from the nature of the cause, which produces such profound disturbances in the nervous system, blood and organs that only a feeble and powerless reaction is possible under the action of so violent a poison.

The action of the poison of yellow fever, during the stage of active febrile excitement, is manifested not only by the intense pain in the head and back, and by the florid congestion of the capillaries of the eyes and skin, and by the loss of appetite and nausea, and by the elevation in the temperature and increase in the frequency of the pulse, but also by the appearance of albumen in the urine, which is rarely absent, and may make its appearance upon the first day of the febrile excitement.

While it is true that the excretory cells and casts of the urinary tubes appear simultaneously with the albumen in the urine, I have also shown by careful analyses of the urine, that the presence of this constituent of the blood in the urine, is not to be referred wholly to acute desquamation of the excretory cells and tubes of the kidney, nor to mere capillary congestion similar in all respects to that in other organs, but equally to the chemic change effected by the poison of yellow fever upon the albumen of the blood.

I have shown by continuous observation and analysis, during different stages of the disease, that the albumen appears in large amounts in cases where there is apparently no failure in the excretory action of the kidneys, and in which the urine was abundant and loaded with urea and other constituents of the urine, and also with bile. While the presence of the blood corpuscles and coloring matters of the blood may be determined by chemic and microscopic investigation of the urine in some cases of yellow fever, at the same time the presence of these constituents of the blood are not uniform and characteristic, as in the cases of that form of severe malarial fever, which, in our Southern States, is now known as malarial hematuria.

In this latter disease the casts and excretory cells of the kidney tubes may be present in considerable numbers, but only that amount of albumen is present which would correspond to the colored blood corpuscles and coloring matters of the blood escaping at the same time.

In yellow fever, on the other hand, even when blood appears in the urine, the albumen is vastly more abundant than the colored blood corpuscles.

The congestion, so marked upon the mucous membrane of the eyes, and upon the breasts and arms, in some cases, a "subcutaneous rash," extends also to the internal organs, and is best observed in the injected nares, crimson lips, scarlet tip and edges of the tongue, and brilliant congested fauces. The fact that the examination of the congested throat causes nausea and retching, when the tongue is pressed down by the finger or spatula, is of importance as a diagnostic sign; and Blair affirmed that when marked congestion is not observed in the eyes and nares and throat in yellow fever, it will be observed by means of the speculum, in the mucous membrane of the anus and vagina. This intense capillary congestion not only characterizes the mucous membrane of the stomach in yellow fever, but it is even manifested in the pericardium after death.

4. The symptoms manifested during the stage of depression and exhaustion, in which the depressing effects of the preceding changes of the blood and organs induced by the action of the febrile poison and its products are manifold. In cases which terminated favorably, the deleterious substances generated during the stage of febrile excitement are gradually eliminated, and the blood purified and relieved of the altered albuminoid and fibrinous elements, and the organs especially affected by the poison, as the heart, liver and kidneys resume their healthy action.

In cases which terminate fatally, in this stage, the issue appears to be determined mainly by the profound alterations of the blood, the failure of the heart, from acute fatty degeneration, and suppression of the action of the liver and kidneys, and the consequent retention in the blood of urea and bile, the poisonous action of the constituents of the urine and bile upon the blood, and nervous and muscular systems and gastro-intestinal mucous membrane.

There appears to be no just ground for the division of the fourth stage, in accordance with the presence or absence of hemorrhage, jaundice, uremic poisoning and convulsions, for these are simply aggravations of the preceding symptoms, and must be regarded as the maximum phenomena of this stage. Neither is this stage to be regarded as similar to, or identical with the intermission or remission of malarial fever, and as an abortive attempt at the repetition of the febrile stage, in which the disease ends or aborts from adynamic incompetency to carry out the phenomena.

These stages are not of uniform duration, and there may be marked diversity in the manifestations of the symptoms in individual cases, some cases presenting, apparently, but the one stage of febrile excitement, while others, overwhelmed as it were by the poison at once, manifest only the symptoms of blood poisoning, arrest of the action of the heart and nervous system and hopeless prostration.

In mild cases, especially in children, the disease may be so slight as scarcely to attract attention, and convalescence may be established in two or three days; in many cases the stage of febrile excitement continues from two to six days, rarely extending beyond three or four days, and the patient may pass almost imperceptibly into convalescence during the stage of depression; in the severest cases the patient may be carried off in two or three days and die with an elevated temperature; while in other severe cases, which end fatally either from hemorrhage from the stomach and bowels, or from urinary suppression, or from both causes, or from general prostration of the system, derangement of the blood and perverted nutrition characterized by a low adynamic fever, and abscess of the parotids, and the revival of old complaints, and the supervention of some other disease, as malarial fever, the fourth stage may be of long duration, extending over weeks and even months.

It is, however, true as a general rule, that convalescence from yellow fever is comparatively rapid, and in striking contrast with the slow and tedious convalescence of malarial fever. In some cases vomiting may be the preëminent symptom, in others, hemorrhage from the stomach and bowels, or from the gums, eyes, and ears, indicating great alteration of the blood, and especially of the fibrinous element; and in others, cerebral symptoms; but all these symptoms may be referred to certain definite results of the action of the poison of yellow fever.

Yellow fever, according to this view, partakes of the nature of a *continued pestilential fever*, presenting two well-defined stages: The first characterized by *active chemic change in the blood and organs*, attended with elevation of temperature, aberration of nervous phenomena, which may constitute the entire malady, and prove fatal in a manner similar to the infectious fever of smallpox; and the other, a *stage of depression induced both by the sedative action of the febrile poison and by the profound changes excited in the constitution of the blood, and in certain organs, as the heart, liver, and kidneys, and by the direct sedative and poisonous action of the excrementitious matters* retained in the blood, in consequence of the failure of action in the liver and kidneys, and by the arrest and perversion of digestion in consequence of the direct action of the yellow fever poison, in causing perverted nervous action of capillary congestion and active desquamation of the secretory cells of the stomach, and in consequence of the elimination of certain constituents from the blood, as the urea and carbonate of ammonia.

While the phenomena of yellow fever are well marked, and different from those of other diseases, at the same time the duration of the disease and the intensity of the symptoms will depend in each case upon the condition of the system at the time of the introduction of the poison, the various agencies to which the system is subjected during the progress of the disease, as the supply of pure air in comfortable, well-constructed and thoroughly ventilated rooms, or of impure air in filthy, damp and crowded habitations, and in filthy and crowded ships, the diet, medical treatment, climate (whether hot and dry or hot and moist, uniform or subject to great and sudden elevations and depressions of temperature, malarious or unmalarious); but the duration of the disease and the character and intensity of the symptoms will especially depend upon the extent to which the blood and organs have been involved and altered by the changes excited by the poison.

The various manifestations, as the capillary congestion, depression of the action of the heart, delirium, coma, and convulsions, vomiting, hemorrhage, and urinary suppression, uremic poisoning and jaundice, with biliary poisoning, may all be referred to the action of the poison producing the disease, and should not be erected into distinct types of disease. And even in those sudden and appalling cases in which there is such a modification of symptoms between the stage of active febrile excitement and that of active depression as to produce a deceitful sense of security, in both the medical attendant and patient, in which period of apparent calm and convalescence the patient asks for food, proclaims himself entirely restored to health, and insists upon getting up and walking about, the sudden fatal issue which astounds the friends of the patient, as well as his medical attendant, has been silently but surely induced by the failure of the kidneys to eliminate the poisonous materials from the blood, and the fatal mischief may have been working for hours and days, unobserved and unrecorded by the medical attendant, who has neglected to examine carefully, at stated intervals, the amount and character of the urinary excretion, through which the deleterious products of the poison are mainly eliminated.

While it is true, that few cases of yellow fever resemble each other exactly, some cases being characterized throughout by symptoms of excitement, and others by prostration; in some cases the cerebral symptoms being most marked, in others hemorrhage from the stomach and bowels constituting such prominent symptoms as to give a distinctive feature to the disease; at the same time there appears to be no just ground for the division of the disease into such distinctive types as would lead to the supposition that the cause might differ in different cases, and that the phenomena might not all be referred to the action and results of a definite cause.

The action of the yellow fever poison is the same in all cases, whether mild or severe; the progress and termination of each case, as well as the manifestation of the various symptoms depending upon the extent of the action of the poison, the condition of the system at the time of its introduction, the peculiarities of the constitution, and the supervention of other diseases.

In a word, the action of the yellow fever poison is distinct, and the disease is characterized by definite manifestations, just as each species of plants and animals manifests distinctive laws of development and growth and physical organization, which may be the subject of investigation and accurate description; and while the laws may be modified in their manifestation by varied extraneous causes, the grand distinctive characters are maintained, and may be propagated from age to age.

Yellow fever is a self-limited disease; it runs a definite course and then ceases. When once fairly established in the human system, yellow fever can not be arrested by drugs. On the other hand, it is well established that paroxysmal malarial fever may be arrested and cured by bark and its active principle quinia. Yellow fever may be conducted to a successful issue by the attentive and careful physician, but it can not be arrested.

PERIOD OF INCUBATION OF YELLOW FEVER.

The period between the exposure to the "infected atmosphere" and the manifestation of yellow fever is not of fixed duration, but varies with different individuals; some systems resist for long periods the morbific agents, while, on the other hand, healthy individuals coming into an infected city have been attacked within thirty-six hours. A few hours' exposure to the atmosphere of the city in which yellow fever is endemic may be sufficient for the introduction of the poison into the system. This statement is sustained by the following observations:

Case 3.—A stout, healthy young gentleman, of florid complexion, sanguine temperament, and active habits, resided in a healthy locality in Liberty County, Georgia, thirty miles from Savannah. In the month of September, 1854, while the yellow fever was prevailing, he drove in his carriage into the city, and visited the house in which one of his near relatives had died a few days before with well-marked yellow fever accompanied with black vomit. Entering the city at midday, he remained about four hours (just long enough to secure some business papers relating to the estate of his deceased relative), and returned immediately into the country. In the course of one week this stout young man, in the full vigor of health, was seized with yellow fever, and died with black vomit after an illness of four days. After death, the body presented a deep yellow color, and the dependent portions were mottled from the unequal and excessive capillary congestion. The exposure of a few hours to the atmosphere of the city was in this case sufficient for the introduction of the poison. On the other hand, many individuals living in the infected cities, and holding daily intercourse with the sick, do not manifest symptoms of the disease until near the close of the epidemic; while others, equally exposed, pass through many epidemics without even manifesting a sufficient amount of febrile derangement to require medical treatment. This latter remark applies not only to the natives of southern cities, but also to residents of northern latitudes, who remove to those localities where yellow fever prevails after they have attained their full growth. The period of incubation appears to vary in different epidemics, and among different individuals during the same epidemic, and may extend from twenty-four hours to weeks and even months. In this respect yellow fever is allied to those diseases which are supposed to arise from malarial effluvia, and differs from those well-defined contagious diseases in which the period of latency is well-marked and of definite duration.

During the prosecution of my investigations in yellow fever, notwithstanding frequent contact with the sick and dead, and exposure to the exhalations from the bodies of yellow fever subjects and from the black vomit, excretions, and blood and organs subjected to chemic analysis, I have never experienced well-marked symptoms of this disease until the great epidemic of 1878; although at times, when most exposed to such exhalations, I have suffered with slight fever, loss of appetite, and nausea (these symptoms, however, were not of sufficient severity to cause the intermission of my labors); and such observations have led me to the belief that under certain circumstances the poison of yellow fever may pass through the system, producing slight derangement, without inducing the disease itself.

During the prosecution of similar investigations in smallpox, I have in like manner felt the depressing influence of the poison without manifesting well-marked symptoms of this disease; and upon one occasion, after exposure to the smallpox poison in the crowded tents of a Confederate military smallpox hospital, and after performing a protracted *post-mortem* upon the body of a Confederate soldier who had died with confluent smallpox, I suffered with fever, intense pain in the back and head, and injection of the capillaries of the skin. These symptoms disappeared, however, at the end of three days. It was evident that the protective influence of the vaccine virus introduced into my arm twenty-five years before was sufficient to cause or allow the passage of the smallpox poison in large amount through the system, with only a partial and mild manifestation of some of the symptoms usual in those unprotected either by vaccination or by a previous attack of smallpox.

In the case of the yellow fever poison, there was no known state of the system artificially produced, as in the case of the smallpox poison, which would account for the elimination of the poison; although that such an elimination must have taken place was manifest from the peculiar train of symptoms attending prolonged exposure to the exhalations of the sick and dead.

Of the various students numbering over one hundred, and many of them fresh from the country, who have observed cases of yellow fever in my wards in the Charity Hospital and witnessed *post-mortem* examinations in the dead house, and chemic and microscopic investigations in my laboratory during the autumnal months of 1870, 1871, 1872, 1873, 1874 and 1875 in New Orleans, only two were attacked with yellow fever. In none of the years specified did yellow fever prevail as an epidemic in the section of the city in which the Charity Hospital is situated. In only a few instances in 1873, did the disease originate among the patients in the wards, who had not been exposed to the infected localities in the city.

There are many persons in the city of New Orleans, who have nursed hundreds of yellow fever patients, stood over them, rubbed and bathed them, inhaled the atmosphere of sick rooms for months, and even washed and shrouded the dead, without ever having a day's sickness. The late Mr. Maybin, a native of Philadelphia, who was distinguished for his devotion to the poor, sick and suffering, informed me that he had passed through all the great epidemics of New Orleans, for a period of over fifty years, including the great epidemics of 1839, 1843, 1844, 1848, 1853, 1854, 1855, 1859, and 1867, without having even a symptom of the disease.

In 1853, many nurses in the Charity Hospital, when two hundred or more died per week, escaped the fever. In the same year, Mr. Logez, one of the carpenters in the Charity Hospital, worked hard all summer, within a few feet of the dead-house where fifteen or twenty bodies were often lying at once awaiting burial, and emitting a nauseating stench, and did not take the disease. Six other carpenters who assisted him took the disease and died. Some of them made the very coffins in which they were buried. The physicians of the Charity Hospital who made careful and thorough *post-mortem* examinations of the bodies in the months of July and August, when the epidemic was at its height, and the bodies in a state of rapid decomposition and offensive, escaped infection or contagion. Over two thousand deaths from yellow fever occurred in the Charity Hospital during the fatal epidemic of 1853. (History of Yellow Fever in New Orleans, 1853, p. 38.)

In *La Republica* of Buenos Ayres, a statement occurs that during the epidemic of 1871 when, according to this paper 17,084 deaths occurred, 350 grave-diggers escaped the fever. The cemeteries are situated on the outskirts of the population. Coffins were exposed for many hours before being interred, from the press of work. (*Medical Times and Gazette*, August 19, 1871, p. 216.)

According to Dr. Daniel Blair, of British Guiana, in some cases the patients had arrived in this colony and in two weeks died of yellow fever, having been attacked within the first twenty-four hours after arrival. Dr. Blair records the following illustrations:

> Miss R. left this colony with her mother and sister in one of Booker Brothers' ships, having been exposed to river influence only the one night previous to departure. All were in good health on embarkation. After being at sea fourteen days they all sickened; Miss R., who had been far advanced in phthisis, died of yellow fever.
>
> Mr. Bramisch had been exposed to the malarial locality of Water Street, at Walmsley and Bowes on Friday. He left town the same day, and sickened with yellow fever on Sunday.
>
> Dr. Reid's exposure and interval between exposure and attack was the same.
>
> Mr. Bolton, of the *Rapid*, was only one night in the malarial district, and within twenty-four hours after proceeding to Sand Hills, was seized with the epidemic.

Thus it would appear that the period of incubation varies, and that some systems can for a longer period throw off the morbific agency; while with others, exposure the most casual and temporary produces immediate effects. A case occurred in the Seamans Hospital, wherein the period of incubation seemed extended to four months being kept latent probably by the phthisis under which the patient was suffering.

Patients previously suffering from intermittent fever, dropsy, iodinism (coryza from iodid of potassium), and ptyalism have had the epidemic disease engrafted thereon; peritonitis and pneumonia failed to exclude the invasion, and the most inveterate lichen tropicus. It has added itself to delirium tremens and lead colic; it has supervened during the flow of the catamenia. Patients under treatment for anemia, and intermittent fever, sarcocele, scald, diabetes and flesh wounds, fractures, rheumatism, erysipelas, ophthalmia, syphilis, strictures, ulcers, eczema, mosquito wounds, whooping cough, and phthisis, have been attacked and often fatally by yellow fever.

When yellow fever became a parasitic disease, the symptoms were considerably modified at first, but the epidemic disease ultimately absorbed all the other symptoms.

In the epidemics observed by Dr. Blair in British Guiana, the invasion sometimes began with malaria of several days duration. Sometimes this formative stage showed itself in diarrhea; in a few cases it began with apoplectic or paralytic symptoms. Sometimes if the treatment was early adopted, or the patient was suffering from another malady, reverberations, repulse and relapse were observed in the first stage, thus abnormally extending its period. About one-half of the normal cases were so sudden and phenomenal in the seizure that the exact hour of the attack could be precisely ascertained. The hours of 6 A.M. and 6 P.M. when, in the latitude of Georgetown the most violent atmospheric disturbances of the day occur, were most favorable to the seizure.

The average duration of an attack of the epidemics of British Guiana, ending in convalescence, estimated from 1,158 cases of the *graver* form, and 428 cases of the *milder* form, was 6.34 days for the former and 5.35 for the latter. The average duration of a fatal attack, estimated from 404 cases, was 7.08 days. In these estimates, the day of attack and the day of death, or of convalescence, are reckoned each one day. Although the maximum and minimum days of duration differed widely from this average, those of the *graver* being 23 and 2, and of the *milder* 13 and 2, and those of the 2,472, yet the vast majority of cases fall in closely with the averages. This coincidence happened always more particularly when the disease was persistent, and the epidemic pulsations most intense. The average, therefore may be assumed as the law of duration, of each class respectively.

According to Dr. Blair, intemperance was occasionally a predisposing cause, by recklessness of exposure; but abstinence, as shown on board the American "teetotal" ships, was no protection. It seemed at the time as if those resident in the infected districts circulated the poison habitually through their systems; that old residents had in an eminent degree the power of eliminating it, and keeping its presence latent—had a tolerance of it; but that newcomers, and particularly those of florid complexions and rigid fiber, were constantly, in reference to the presence of the virus, in a state of tottering equilibrium; so that in them the slightest unfavorable impulse to that balance—the lowering of the vital powers by fatigue, the suppression of any of the preparatory secretions, a shock to either of the nervous centers, or the depressing emotions—were sufficient to excite the latent poisons. (Some Account of the Late Yellow Fever Epidemic of British Guiana, Third Edition, 1852, pp. 60, 61, 69, 70, 72, 74.)

According to Alexander Humboldt, the farm of l'Encero, near Vera Cruz, elevated 3,043 feet above the level of the ocean, is the superior limit of the *vomito.* The Mexican oaks descend no farther than that place, being unable to vegetate in a heat sufficient to develop the germ of yellow fever. Individuals born and brought up at Vera Cruz are not subject to this disease; and it is the same with the inhabitants of Havana, who do not quit their country; but merchants born in the island of Cuba, or who have inhabited it for a great number of years are attacked with the *vomito pireto* when their affairs oblige them to visit this part of Vera Cruz during the months of August and September, when the epidemic is at its height. In the same manner, Spanish Mexicans, natives of Vera Cruz, have been seen to fall victims to the *vomito* at Havana, Jamaica or the United States.

The *vomito* is not regarded as contagious at Vera Cruz. In most countries, the common people consider many diseases as contagious which are of a very different character; but popular opinion in Mexico has never interdicted the stranger not seasoned to the climate from approaching the beds of those attacked by the *vomito.*

The whites and mestizoes who inhabit the interior tableland of Mexico, of which the mean temperature is 60.8 degrees and 62.6 degrees F., and where the thermometer sometimes falls below the freezing point are more liable to contract the *vomito* when they descend from l'Encero to the Plan del Rio, and from there to la Antigua and the port of Vera Cruz. than the Europeans or inhabitants of the United States who come by sea; the latter passing by degrees into the southern latitudes are gradually prepared for the great heats which they experience on landing; but the Spanish Mexicans, on the other hand, change suddenly their climate when in the space of a few hours they are transported from the temperate region to the torrid zone. The mortality is very great, especially among two classes of men very different in their habits and modes of living; the muleteers (*arrieros*) who are exposed to extraordinary fatigues in descending with their beasts of burden, by tortuous roads like those of St. Gothard, and the recruits destined to complete the garrison of Vera Cruz. Every imaginable care has been bestowed on these unfortunate young men, born on the Mexican tableland at Guanaxuato, Toluca, or Pueblo, for the purpose of preserving them from the deleterious miasmata of the coast, but without success; they have been left for several weeks at Xalapa, to season them gradually to a higher temperature; they have descended on horseback, or by night to Vera Cruz, that they might not be exposed to the sun in crossing the arid plains of Antigua; they have been lodged at Vera Cruz in well-aired apartments; but it has never yet been observed that they were attacked with the yellow fever with less rapidity and violence than the soldiers for whom these precautions had not been taken.

In the season when the *vomito* rages with great violence the shortest stay at Vera Cruz, or in the atmosphere which surrounds the city, is sufficient to communicate the disease to persons not seasoned to the climate.

The inhabitants of the City of Mexico when they propose to sail for Europe, dreading the insalubrity of the coast, generally remain at Xalapa till the moment of the departure of their vessel. They set out on their journey in the cool of the night or cross Vera Cruz in a litter, to embark in the boat which awaits them at the Mole; and yet these precautions are sometimes useless, and it happens that these very persons are the only passengers who sink under the *vomito*, during the first days of the passage. It might be admitted that in this case, the disease has been contracted on board the vessel, which remained in the port of Vera Cruz, and which contained deleterious miasmata; but the celerity of the infection is more incontestably proved, by the frequent examples of the better class of Europeans, dead of the *vomito* though on arriving at the Mole, they may have found litters ready to begin the journey to Perote.

Such facts may be held by some to sustain the doctrine that yellow fever is contagious, under all the zones; while those who hold that it is not communicable by immediate contact with the sick and dead, admit that the atmosphere of Vera Cruz contains putrid emanations, which if breathed for the shortest space of time, introduce disorder into all the vital functions.

The most part of the Europeans newly landed feel during their stay at Vera Cruz the first symptoms of the *vomito*, which is announced by a pain in the lumbar regions, by the yellow covering of the conjunctiva, and by signs of congestion toward the head. In some individuals it only declares itself when they arrive at Xalapa, or in the mountains of La Pileta, in the region of pines and oaks, at from 5,248 to 5,904 feet above the level of the ocean. Those who have long resided at Xalapa, deem themselves able to foretell from the features of the travelers who ascend from the coast to the tableland of the interior, whether without their being sensible of it themselves, they contain within them the germ of the disease. Dejection and fever increase the predisposition of the organs to receive the impression of the miasmata; and these same causes render the commencement of the yellow fever more violent when the patient is imprudently informed of the danger of his situation. In illustration of the preceding statement, Alexander Humboldt cites the following interesting instance:

A person with whom he was on terms of intimate friendship, during his stay at Mexico, had passed only a very short time at Vera Cruz, on his first voyage from Europe to America. He arrived at Xalapa without feeling any sensation indicative of the dangerous state in which he was immediately to be. "You will have the *vomito* this evening," said an Indian barber, gravely to him while he lathered his face; "the soap dries as fast as I put it on; that is a never-failing sign, and for the twenty years that I have been in the practice of shaving the *chapetons*, who pass through this town on their way to Mexico, out of every five, three have died." This sentence of death made a strong impression on the traveler. It was in vain to represent to the Indian that his calculation was exaggerated, and that a great heat of the skin does not prove this infection; the barber persisted in his prognostic, and in reality the disease declared itself a few hours afterward, and the traveler already on his way to Perote, was obliged to be transported to Xalapa, where he nearly fell a victim to the violence of the *vomito*. (Political Essay on the Kingdom of New Spain, London, 1811, Vol. IV. pp. 170–178.)

In regard to the epidemic of Gibraltar in 1804, Sir W. Pym[3] states that the *fourth* day was generally the time the contagion seemed to require to show itself; and several instances occurred of strangers who were attacked on the second and third day after their landing at Gibraltar. From the fact that persons were seized on the first day of their landing at Gibraltar, while persons who fled from the garrison were taken ill on the coast of Portugal many days after, induced Mr. Amiel to believe that the disease may be excited into action at any time from the first moment of exposure to a period of fifteen or twenty days. The following statement by Wm. Fergusson, in his "Notes and Recollections," would even show that the disease may be developed in less than twenty-four hours after exposure to the infected atmosphere. At Mark's Hill, "it was the duty of the white troops in both forts to take the guard and duties of the dock yards among the marshes below; and so pestiferous was the atmosphere, that it often occurred to a well seasoned soldier mounting the night-guard in perfect health, to be seized with furious delirium while standing sentry, and when carried back to his barracks on Mark's Hill, to expire in all the horrors of the black vomit, within less than thirty-six hours of the first attack."

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[3] Observations on the bulam fever which has of late prevailed in the West Indies, on the coast of North America, Gibraltar, Cadiz and other parts of Spain, etc. S. tr., London, 1815, p. 24.

Drs. Pausit, Bally, and Francois regarded the period of incubation at Barcelona in 1821, as not exceeding twenty-four hours, or three days at the most; Aujula from twenty-four to thirty-six hours, sometimes from two to seven days, and even as late as from thirty to fifty days.[4] According to Dr. Harrison, persons who arrived in New Orleans during an epidemic, from the healthiest region, even by the ocean, are subject to attack on the third to the sixth day after their arrival; and Dr. Merrill[5] states that at the Bay St. Louis in 1820, several persons who arrived while the disease was at its height were attacked in twenty-four hours.

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[4] Pausit.—Observations Sur la Fièvre Jaune fales à Cadiz en 1819, etc.

[5] New Orleans Medical and Surgical Journal, March, 1847, p. 569.
Yellow Fever, by R. La Roche-Mer, Vol. I, p. 510.

Dr. D. O. Saunders, of Memphis, placed the incubation period of the fever of 1873, between twenty-four hours and sixteen days, having seen one case where a gentleman who had been absent all summer was attacked in twenty-four hours after his return, and another who was away from the city was attacked in sixteen days after leaving New Orleans. 1874, p. 800.

The authority of various other observers, as Lind, John Hunter, R. Jackson, Wallace, Riseuno, Moreau de Jennès, Rush, Townsend, Chisholm, Bryson, might be adduced to sustain the proposition already sufficiently illustrated by the authorities quoted, that the period of incubation of yellow fever is of variable duration. From the preceding facts, the following conclusions may be drawn:

1. Yellow fever differs from such contagious diseases as smallpox, measles and scarlet fever, in the variable duration of the period of incubation.

2. The sudden seizure of many cases after a few hours exposure to the infected atmosphere, indicates the existence in the air of a potent and specific poison, which is most probably introduced through the lungs into the blood.

3. The phenomena of yellow fever in those suddenly exposed to the atmosphere of an infected locality can not be referred to any physical changes of the temperature and electrical condition of the surrounding atmosphere, but must be referred to the action of a specific poison.

4. The propagation of yellow fever from the infected atmosphere of an infected vessel in a healthy city; the communication of the yellow fever from person to person; the spread of the disease from an infected point in a city, however large, over extended areas of said city; and the sudden cessation of epidemics of yellow fever by cold sufficient to produce frost and ice. These and similar facts indicate that the poison of yellow fever is a living germ of animal or vegetable nature or origin.

CHAPTER II.

COMMUNICATION OF YELLOW FEVER THROUGH THE MOTHER TO THE FETUS IN UTERO; WITH OBSERVATIONS UPON THE COMMUNICATION OF SMALLPOX TO THE FETUS.

It is possible for yellow fever to be communicated through the mother to the fetus in utero.

I have had opportunity of illustrating this proposition by the following observation:

Case 4.—Illustrating the communication of yellow fever through the mother to the fetus in utero. Mary Heitman entered Charity Hospital, New Orleans, Louisiana, Oct. 1, 1871, with fever, and intense pain in head and back. On October 4 (third day of disease) patient presents all the symptoms of yellow fever; conjunctiva jaundiced, eyes injected, capillaries of mouth and gums congested, capillary circulation sluggish, surfaces of extremities mottled, nausea and vomiting. Patient suffering with uterine hemorrhage.

October 5 (fourth day of disease) the patient gave birth to a still-born fetus, apparently about four months old. The skin of the fetus presented a jaundiced yellow hue, and, upon dissection, the liver of the fetus presented the usual appearance of this organ in yellow fever. On October 6 this woman died, apparently from the immediate depressing effects of hemorrhage from the uterus and stomach. The pathologic lesions were those of yellow fever. The serum from the blood of the cavities of the heart presented the usual golden color, and contained bile. The colored blood corpuscles were altered in their outlines, some being swollen and others crenated. The muscular structures of the heart were yellow and brownish-yellow, softened and loaded with oil globules. The liver presented the characteristic yellow color of this disease, and liver cells were loaded with oil. The kidneys presented a yellow and yellowish-red color, resembling that of the liver and heart. Sections of the kidney, by Valentine's knife, carefully examined under the microscope, revealed the tubuli uriniferi, filled with granular fibroid matter, epithelial cells and oil globules. The urine taken from the bladder after death contained albumen in large amount, and urea in comparatively small amount. Under the microscope the urine was found to contain numerous oil globules, casts of the tubuli uriniferi, filled with yellow granular albuminoid and fibroid matter, and in addition to these, numerous free cells from the tubuli uriniferi.

Louis, in his "Anatomical, Pathological and Therapeutical Remarks on the Yellow Fever of Gibraltar of 1828," records the observation that the uterus of a woman, the wife of a soldier of the Fifty third Regiment, who died with well-marked yellow fever, contained a five or six months' fetus, about whose neck were ecchymoses and whose skin was of a universal wine-lees color. The amniotic fluid appeared to have been colored by bile. In this respect the poison of yellow fever resembles that of certain contagious diseases, as smallpox. Thus instances have been recorded in which the fetus in utero has been attacked with smallpox, in consequence of the mother having been exposed to the contagion, without herself having the disease.

It was long held as a popular opinion, that if persons were insusceptible to the smallpox, either from infection in consequence of repeated exposure, or from inoculation with variolous matter, that they must have undergone the disease previous to their birth. Some medical men, on the contrary, have held that the want of susceptibility in such persons to the infection of the smallpox was owing to a peculiarity of constitution, which resisted the influence of the disease.

In support of the latter opinion, Mr. William Forbes[6] recorded the following case:

Case 5.—Mrs. Tagg, 22 years of age, when in the seventh month of her first pregnancy, had the confluent smallpox; from which, contrary to expectation, she recovered, went her full time, and was delivered of a fine child. Frequent solicitations were made for permission to inoculate the child, which was refused by its parents, from a supposition of its having had the smallpox previously to its birth. However, at the age of 12, the child was inoculated, and had the smallpox in the most satisfactory manner.

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[6] Cases of Smallpox in the Fetus, by William Forbes, Edinburgh Medical and Surgical Journal, Vol. iii, p. 307, 1807.

The two following cases, recorded by Mr. Forbes, on the contrary, tend to prove that those persons who have resisted the variolous infection, in every form, must have undergone the disease when in the fetal state:

Case 6.—Mrs. Alexander, of New Cross, had the smallpox when near the termination of her pregnancy; but the type was so mild that she was capable of pursuing her occupation as a washerwoman, although in daily expectation of being confined. The infant was born with the smallpox upon it.

Case 7.—Margaret, wife of William Crawby, residing at Camberwell, in her infancy had the smallpox severely, to which her countenance bore ample testimony. In March, 1788, she nursed her son Thomas, a child, under the confluent smallpox. She was at that time pregnant of her fifth child, and very near the end of her reckoning. These people being in low circumstances, had but one room, in which the whole family lived and slept. On April 1, at 3 o'clock in the morning, she was delivered of a girl. Three days afterwards the boy Thomas died, and on the same day Mr. Forbes was desired to look at some pimples upon the infant, which proved to be the smallpox, the marks of which she bore upon her face, even after attaining womanhood, and although frequently exposed to the smallpox, never contracted the disease.

The latter case proved that the fetus in utero is liable to the smallpox, from the influence of surrounding infection, although the mother was herself protected by a previous attack of the disease.

Mr. William Rait[7] recorded the following case, similar in some respects, and even more remarkable than that of Margaret's, just quoted:

Case 8.—During the prevalence of smallpox, and while Mr. Rait was attending such cases, a lady, who manifested no symptoms of smallpox, was delivered of a male child, upon whom the smallpox eruption appeared two days after its birth. The disease went through the course of genuine smallpox. Upon inquiry Mr. Rait ascertained from the mother of the lady (grandmother to the child) that the mother was born with the smallpox.

Dr. Laird[8] has described the following case of a pregnant woman, who was seized with smallpox:

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[7] Medical Commentaries, by Andrew Duncan Vol. iii, p. 318, 1789.

[8] Edinburgh Medical and Surgical Journal, Vol. iii, p. 155, 1807.

Case 9.—About the end of August, 1805, she felt the motion of the child till the month of October, and on October 28 she was delivered of a dead child, which was thought to be of six months' growth. On the back, shoulders and side, particularly upon the upper parts of the thigh, where the integuments were perfectly sound, there were several pustular eruptions, with central depressions, strikingly characteristic of the appearances which distinguish smallpox. The fetus was placed in the museum of George's Hospital, and distinctly exhibited the characters of the eruption.

This history confirms the observation that the fetus partakes in many of the diseases of its parent.

Mr. Lynn published in August, 1786, the following decisive instance of smallpox in the womb:

Case 10.—In November, 1785, the wife of Mr. Ere, a coachmaker in Oxford Street, being then in the eighth month of her pregnancy, was seized with vigorous pain in the back, and other febrile symptoms. In two days' time the disease showed itself to be the smallpox, and though the pustules were of the distinct sort, yet they were uncommonly numerous. On the eleventh day they began to turn, and on the twenty-second day her labor took place, which, according to her reckoning, was a fortnight before the regular period; that is, when she was advanced in her pregnancy eight months and two weeks. The child, at the time of its birth, was covered with distinct pustules, all over its body. They did not appear to be full of matter till three days after, at which time I took some of the pus upon a lancet from one of the pustules on the face. With this lancet Dr. Lynn afterwards inoculated, on Dec. 2, 1785, a child of Mr. Charters, in Church Street, in both arms. On the 7th, the inflammation began to appear in each arm, and continued daily increasing, till December 11, when the child sickened, and was affected with all the symptoms which usually precede the eruption. On the 12th the sickness and fever abated, the pustules of the distinct sort of smallpox made their appearance, and the child having regularly gone through the several stages of the distemper, was perfectly well in three weeks.

Dr. Edward Jenner[9] entertained the opinion that the susceptibility to receive the variolous contagion always remains through life, but under various modifications and gradations from that point, when it passes silently and imperceptibly through the constitution (as is frequently the case with cowpox), up to that where it appears in a confluent state, and with such violence as to destroy life.

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[9] Medico-Chirurgical Transactions, Vol. i, London, 1815, Third Ed., pp. 271-277.

It is only under particular circumstances that any proof of the presence of smallpox can be adduced in those cases, in which it passes through the frame without producing eruption, or in any perceptible degree deranging the animal functions.

Such proof, however, as Dr. Jenner clearly exemplified by the following remarkable cases, is afforded by the obvious infection of the fetus before birth, communicated through the mother, herself being already secure from any visible occurrence of the disorder:

Case 11.—Dr. Edward Jenner was requested by Dr. Croft to vaccinate the infant of Mrs. W., a lady in Portland Place. The vaccine fluid, which was inserted fresh from the arm of another infant, produced scarcely any effect, beyond a little efflorescence in the part, which in a few days disappeared. When Dr. Jenner expressed his surprise at this, such an occurrence happening very rarely, Mrs. W. removed his embarrassment by the following narrative:

A few days previous to her confinement she met a very disgusting object, whose face was covered with smallpox. The smell and appearance of the poor creature affected her much at the time, and though she mentioned the circumstance on her return home, she had no idea that her infant could suffer from it, having had the smallpox herself when a child.

During a few days after its birth, the little one seemed quite well, but on the fifth day, it became indisposed, and on the seventh the smallpox appeared.

The pustules, which were few in number, matured completely. Dr. Croft, who attended her, being curious to know the effect of inoculation from one of the pustules, put some of the matter taken from one of them into the hands of a gentleman eminently versed in the practice, which produced the disease correctly. Mrs. W. was not sensible of any indisposition from the exposure, nor had she any appearance of the smallpox.

Another case, similar in its general character to the above, was communicated to Dr. Edward Jenner, by Mr. Henry Gervis, a surgeon of eminence at Ashburton, in Devonshire.

Case 12.—Mr. Gervis says: "The smallpox having appeared in the village of Woolson Green, about three miles from Ashburton, on May 6, 1808, I vaccinated a poor woman, the wife of James Barkwell, who was in the last month of her pregnancy. Her three children had been inoculated the preceding day with variolous matter, by the surgeon who attended the poor of the parish, and who had very properly declined inoculating her also, from her particular situation. I made two punctures in each arm, each of which fortunately succeeded, and she regularly passed the disorder, complaining only on the tenth and eleventh days, when the areola was most extended, as usual. I saw her very frequently during the progress of the disorder, and once or twice after its complete termination. I therefore can speak positively, that during that time she labored under no symptoms but what are connected with the cowpox. From this period she continued perfectly well, and on Saturday the 11th inst., she was delivered of a female child, having at the time of its birth many eruptions on it, bearing much the appearance of smallpox, in the early stages of the disease. This event happened five weeks after her vaccination, and one month after she had been exposed to the variolous infection of her own three children, and that of several other persons in the same village. On the 14th I visited the child again, when I found the eruptions had increased to some thousands, perfectly distinct, and their character well marked. . . . To put the matter beyond all doubt, I armed some lancets with virus, and produced the smallpox, by inoculating with it. In addition to the circumstance of the mother conveying the variolous infection to her unborn child, without feeling any indisposition from its action in her own constitution, I must remark, that there can not be a stronger proof of the efficacy of vaccination than this case afforded."

Dr. Jenner, without producing more examples, of a similar description, though he affirmed that many were before him, observes that a fact, not unlike the preceding, fell under the observation of Dr. Mead, who, in his discourse on the smallpox (Chap. iv., p. 337, 1772), relates the following case:

Case 13.—A certain woman who had formerly had smallpox, and was near her reckoning, attended her husband in the distemper. She went her full time, and was delivered of a dead child. It may be needless to observe that she did not catch it upon this occasion; but the dead body of the infant was a horrid sight, being all covered with the pustules, a manifest sign that it died of the disease before it came into the world.

From such examples, Dr. Jenner[10] concludes that the smallpox virus may affect the human frame, even to its inmost recesses, although apparently secured from its effects, and yet give no evidence of its presence, by exciting any perceptible disorder.

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[10] Medico-Chirurgical Transactions, Vol. i, London, Third Ed., pp. 271-277, 1805.

In 1749, Sir William Watson[11] published the following case:

Case 14.—A woman far advanced in pregnancy, who had labored under the smallpox a long time before, who, during this pregnancy, performed the duty of a nurse to her servant in the natural smallpox; and in a month after this attendance, was brought to bed of a child that had about forty scars upon its body like those from the smallpox. This child (a girl), and her brother, were afterward inoculated at the same time. The brother had inflammation of the parts inoculated, eruptive fever and eruptions, as in the most ordinary smallpox; and the girl, born with the pits on her skin, had inflammation and suppuration in the parts inoculated, in the same manner as her brother, and a general indisposition, as in the undoubted cases of smallpox, but no eruptions.

Sir William Watson concluded that this girl had gone through the smallpox before her birth, and Camper and Van Swieten coincided with him in this conclusion.

It was established by actual experiment, that pregnant women were not fit subjects for inoculation, as the smallpox often caused miscarriage, and what is remarkable, when the disease proved mild to the mother, it was sometimes fatal to the child.

The preceding facts, illustrating the communication of smallpox to the fetus in utero, might be greatly enlarged by other observations, as those of Bartholini, 1657; Mr. Durham, 1713 (Phi. Trans., Vol. xxviii., p. 165); Dr. Mortimer, 1749 (Phil Trans., Vol. xlvi., p. 233); Dr. Mead, 1747, in his Treatise de Variolis; Dr. Rosen von Rosenstein, 1756; Baron Dimsdale, 1766, Treatise on Inoculation; Mr. Wastall, 1776 (Phil. Trans., Vol. lxx., p. 128); Dr. Wright, 1781 (Phil. Trans, Vol. lxxi., p, 372); Dr. Bland, 1781 (*London Medical Journal*, Vol. ii., p. 205); Mr. Roberts, 1784 (*Medical Journal*, Vol. v., p. 399); Dr. Hagarth, Dr. Woodville, Dr. Ford, Mr. Jones, Dr. Hossack; Dr. George Pearson, 1794 (Medical Commentaries, by Andrew Duncan, Vol. ix., Edinburgh, 1795, p. 213); Dr. George Pearson gives some important cases of equal interest with those recorded above; Alexander Munro (Observations on the Different Kinds of Smallpox, Edinburgh, 1818, p. 626).

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[11] Philosophical Transactions, Vol. xlvi, p. 239.

We have thus brought together a number of important observations, scattered through various medical and scientific periodicals, bearing upon the transmission of the poison of smallpox, through systems entirely protected from its influence and demonstrating the existence of the poison in a potential active state, as shown by the excitation of genuine smallpox in the fetus, resting in the womb of the mother, protected by previous inoculation, vaccination and smallpox, *in the hope that the attention of physicians may be directed to analogous inquiries in yellow fever.*

If, therefore, the poison of yellow fever is capable of affecting the fetus in utero, in an analogous manner to the poison of smallpox, we may arrive at the following conclusions, which, if more extended investigation should clearly establish, will explain one mode in which the natives of those cities and localities, as New Orleans, where yellow fever often prevails, obtain an immunity from its attacks:

1. The poison of yellow fever is capable of affecting the fetus in utero.
2. It is probable that the yellow fever poison is capable of affecting the fetus in utero, without inducing the disease in the mother, who has had the disease at some period preceding pregnancy.
3. It is thus rendered probable that a certain number of natives are in this manner rendered insusceptible to the action of the yellow fever poison after birth.

MODIFICATIONS OF THE PHENOMENA OF YELLOW FEVER BY PRECEDING DISEASED STATES OF THE SYSTEM.

The most potent cause of derangement of those who reside within the yellow fever zone is the action of malaria, which not only manifests its effects in the causation of the various forms of intermittents, remittents, pernicious and hemorrhagic paroxysmal fevers, which differ materially in their origin, symptoms and pathology from yellow fever, but also, without the active manifestation of these forms of fever, in the slow destruction of the colored corpuscles, derangement of the liver, enlargement of the spleen, attended with a pale, sallow, sickly hue, infiltration of the cellular tissue, dyspnea, palpitations, derangements of the blood and nervous system, and depressions of the muscular and nervous forces.

While those who have been subjected for long periods to the action of malaria appear to be less liable to yellow fever, it is, however, true that the progress of this disease is modified by the changes induced in the blood and organs by the preceding action of the malarial poison; and the lesions after death from yellow fever differ to a certain extent from those observed in subjects freshly arrived from cold climates without being previously subjected to the action of the marsh miasm. And in every epidemic of yellow fever the malarial influence is so powerful in most of the cities of the tropical, subtropical and temperate regions of North and South America that it is never entirely suspended, and not only in many cases induces characteristic changes before the specific action of the yellow fever poison, but also is frequently engrafted upon the weakened convalescents from yellow fever, thus altering the farther progress of such cases, and inducing changes in the organs entirely different from those characterestic of yellow fever.

In this continuous and preceding action of the malarial or paludal poison, and in the frequent intermingling of the two diseases, we have an explanation of the apparently contradictory statements of observers as to the characteristic symptoms and lesions of yellow fever. It is evident therefore, that no observer is competent to the elucidation of the pathology of yellow fever who is not at the same time familiar with the changes induced by the various forms of paludal fever and malarial poisoning.

If we accept without reserve the doctrine advocated by John Hunter,[12] and ably supported by Joseph Adams,[13]—that "No two actions can take place in the same constitution, or in the same part, or at one and the same time; no two different fevers can exist in the same constitution, no two local diseases in the same part at the same time,"—then the question of the modification of malarial fever by yellow fever, and of the engrafting of the one upon the other, must be definitely settled in the negative.

We have elsewhere shown by an extended discussion[14] of this question that while, when two poisons representing two distinct exanthemic diseases act simultaneously upon the human being, the most obvious pathologic phenomena excited by the poisons will not occur simultaneously, but in succession—the one poison retarding the action of the other; the one producing its cycle of changes, while the other remains *dormant*, as it were, during the action of the first, and immediately after the changes induced by this cease, causing in turn its own distinctive effects—at the same time, it must be admitted that the character and cause of the specific eruptive diseases are greatly modified by such altered states of the constitution as exist in scurvy, scrofula and secondary syphilis. In that class of diseases represented by constitutional syphilis, scurvy and malarial poisoning, the blood is at fault, the nutrition is perverted and the course and products of diseased actions are correspondingly modified.

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[12] Works of John Hunter, edited by James F. Palmer, London, 1827; Vol. ii., Treatise on Venereal Disease, p. 132; Vol. iii., Treatise on the Blood, pp. 3-5; Vol. i., Principles of Surgery, pp. 312, 313.

[13] Observations on Morbid Poisons, Chronic and Acute, London, 1807, second edition, pp. 21-23.

[14] Researches in Spurious Vaccination, or the Abnormal Phenomena accompanying and following Vaccination, in the Confederate Army during the recent American Civil War, 1861-65, pp. 33-59, by Joseph Jones, M.D., formerly Surgeon in the Provisional Army of the Confederate States.

During the recent war, I embraced an opportunity for observing the effects of engrafting smallpox upon patients broken down by exposure, privation and the exhausting effect of hospital gangrene and pyemia.

In the month of September, 1864, smallpox spread from the ward devoted to the treatment of this disease to the Empire Hospital, which had been filled with cases of hospital gangrene and pyemia gathered from the general hospitals attached to the Confederate Army operating in and around Atlanta. The smallpox ward was situated in a pine grove about three hundred yards in the rear of the gangrene hospital; and without doubt communication was kept up between the different wards. Several of the nurses of the gangrene hospital were first attacked, and the disease appeared to have been communicated from them to the patients under their charge who were suffering with hospital gangrene and pyemia.

The following case arrested my attention as clearly illustrating the engrafting of smallpox upon a system reduced by hospital gangrene:

Case 15.—J. S. J., Company K, Nineteenth Alabama Regiment, age 21, wounded at the siege of Atlanta on July 22, 1864. Gunshot wound of right leg, which was amputated on the field of battle at the lower third of the thigh. Hospital gangrene attacked the stump, and the patient was admitted into the Empire Hospital on September 22. Nitric acid arrested the progress of the gangrene for a time, but it returned again, and the strength of the patient was gradually reduced by the absorption of the gangrenous matter and from an exhausting diarrhea. On November 2, while the patient was exceedingly feeble and nervous, and manifesting the symptoms of pyemia—viz., chills, icterus and vomiting of dark green matters—the eruption of varioloid made its appearance. The pustules, which were in considerable numbers, but small in size, progressed regularly, and on November 6 presented all the characteristics of the true variolous eruption, with the round form and umbilicated center. This patient had a good vaccination scar, and the smallness of the pustules appeared to be due to the fact that the system was partially protected by vaccination. The patient died during the night of the 6th, apparently from the effects of the gangrene, pyemia and exhausting diarrhea.

None of the pustules in the preceding case presented any appearance of hospital gangrene; and, so far as I was able to learn, both during my investigation and subsequently, no case occurred in the gangrene hospital in which moist gangrene attacked the eruption of smallpox or varioloid.

It was clearly established that smallpox would attack patients suffering with gangrene, and even pyemia.

Case 16.—In an interesting case which occurred in my private practice immediately after the close of the recent struggle for the rights of self-government in the Southern States, acute dysentery of a severe form was superseded by typhoid fever, which was well marked in its symptoms and progress by low muttering delirium, diarrhea, tympanites, lenticular rose-colored spots, and painful enlargements of the parotid glands. As soon as well-marked typhoid symptoms were manifested, all the symptoms of acute dysentery vanished, and the patient passed without pain or straining the ordinary bilious stools of typhoid fever, and the smallest doses of purgative medicine, either mercurial or saline, acted with great and dangerous violence, producing copious "pea-soup" stools.

The patient passed safely through the typhoid fever, and even the parotid glands subsided without suppuration; but in the third week after the appearance of the typhoid fever, when the patient was apparently doing well, the dysentery returned with severe straining, and the bloody and mucoid discharges of acute dysentery, and the patient died in consequence of the return of the original disease.

I have also observed that typhoid fever was often engrafted upon malarial fever among the Confederate troops serving in malarious districts, and that during its active stages it presented the usual continuous fever, with cerebral disturbance, agitation of the muscles, tympanites, diarrhea, and lenticular rose-colored spots. As soon as the typhoid fever was established, all signs of periodicity disappeared, and the torpid liver of malarial fever became active, the sallow hue of malarial fever became clear from the increased action of the liver, the torpid bowels loose and tympanitic; and after the disappearance of the symptoms characteristic of typhoid fever, the original malarious periodic fever reappeared, with its cold and hot stages recurring at regular intervals, and inducing torpor of the liver and bowels, and the sallow malarial hue.

As far as my observation extends, in like manner, when yellow fever is engrafted upon a system previously under the influence of the malarial poison, it establishes its own peculiar train of symptoms, distinct from those of malarial fever, and may be clearly recognized. When, however, the cycle of changes excited by the yellow fever poison has been completed, then the malarial poison may excite its characteristic recurring paroxysms, and change the yellow fatty liver of yellow fever into the dark slate-and-bronze liver of malarial fever, loaded with dark pigment granules.

While we know but little with reference to the concurrent action of the poisons of these two diseases upon the same system, and know nothing whatever of the state and mode of existence of that poison which "lies dormant" while the other is acting, at the same time we shall endeavor to illustrate the differences and relations of the symptoms and lesions by actual observations.

RELATIONS OF YELLOW FEVER, MALARIAL FEVER, AND MALARIAL HEMATURIA.

I have endeavored by careful observation of the various symptoms, by analysis of the blood secretions and excretions, and by careful examination of the pathologic lesions after death, to unravel the complicated chain of phenomena characteristic of yellow fever and other diseases; and while many facts are unexplained, and much remains to be investigated, we feel assured that the labors which we have pursued unremittingly during the past twenty-two years have been at least in the right direction.

The malarial poison induces profound alterations in the constituents of the blood. Under its action, as I have demonstrated by the first series of investigations (Trans. AMERICAN MEDICAL ASSOCIATION, 1859), colored blood corpuscles are more rapidly and to a greater extent destroyed than in any other disease; the fibrin is diminished and altered in quantity and quality; the albumen is in like manner diminished; the extractive and coloring matters of the blood are frequently increased. The unhealthy hue of the complexion in malarial fever appears to be due to the destruction of the colored blood corpuscles and the presence of coloring matter in the blood; the deposit of pigmentary matter, and the failure of the liver to separate fully the coloring matter of the bile.

In yellow fever there is no marked or uniform destruction of the colored blood corpuscles. The fibrin is diminished, and to a much greater extent than in malarial fever. There is no tendency to the formation of fibrinous concretions in the cavities of the heart in yellow fever, while the formation of such laminated fibrinous clots is common in malarial fever, and in some cases is the manifest cause of death.

Malaria, by its effects in inducing sudden congestions, and by its depressing effects upon the heart and upon the general and capillary circulation, and by its potent action on both the sympathetic and cerebro-spinal system of nerves, tends to promote the formation of heart clots, although there is an actual diminution of the fibrin in the blood during malarial fever. In view of the rapid, feeble, intermittent pulse; disturbed, panting respiration; feeble, rapid, fluttering action of the heart; cold extremities, exhaustion of the muscular forces, stupor, wandering of the intellect, inability to control the muscles and acts of excretion; in view of the sudden onset of all the symptoms in malarial fever; in view of the observations which we have carefully recorded heretofore upon the lesions characteristic of malarial fever, we are justified in asserting that the fibrinous elements of the blood may be deposited in the heart and blood vessels during life in malarial fever, and not only give rise to distinct phenomena, but cause death in cases which otherwise would not have terminated fatally.

It is worthy of observation that in pyemia and in malarial fever, in both of which diseases there is a more rapid destruction of the colored blood corpuscles than in any other class of diseases, *chills* should characterize both affections, and form the most marked symptom. If such facts do not point out the nature of the cause of malarial fever, they at least sustain the belief that this disease, like pyemia and yellow fever, is due to the action of a special poison, and not to mere variations of climate and changes of moisture and temperature.

The rapid destruction of the colored blood corpuscles in malarial fever is evident, not only by a comparison of the constitution of the blood in this disease with that of yellow fever, but also by the presence of a larger amount of coloring matter in the urine. As a general rule, the graver the case of malarial fever the more deeply colored is the urine; while on the other hand, the reverse is the case with yellow fever. The coloring matter of the urine in yellow fever is due to a great extent to the retention of the biliary matters in the blood and the failure of the action of the liver; while on the other hand, the deep red and reddish-brown and orange-colored pigments of the urine of malarial fever appear to be derived chiefly from the colored blood corpuscles,

While the presence of the coloring matter in large amount in the urine of malarial fever may be dependent in part upon some imperfection in the excretion of carbon by those organs whose special function it is to eliminate this element from the blood, as the liver and lungs, and may, as has been observed by Golding Bird, be connected with some functional or organic mischief of the liver and spleen or some other organ connected with the portal circu, lation; at the same time, from a careful consideration of the accompanying symptoms and subsequent *post-mortem* revelations, we have been led to the belief that in malarial fever the pigment is derived chiefly from the coloring matter of the blood cells, and that its amount may be taken as an index or measure of their destruction. This would be true, whether it comes at once from the blood corpuscles by changes taking place in the mass of the circulating fluid, or by the destruction of the blood corpuscles in the liver and spleen. Certain it is that this pigment is not found in the kidneys, and does not accompany diseases of the kidneys; nor is it thrown off under the action of organic medicines and compounds, drastics and purgative salts, which irritate and even cause disease of the intestines and kidneys. Even tincture of cantharides, when given in such large doses as to cause albuminuria and even blood to appear in the urine, does not cause such pigments as purpurine (Bird), uryoërythrin (Heller), or urohematin urophæin to appear in the urine. In those cases of yellow fever in which we have the greatest irritation of the kidneys, or rather in which there is the greatest structural alteration of these organs, will be found, as a general rule, the lightest colored urine. On the other hand, poisonous metallic salts, which derange the constitution of the colored blood corpuscles, and interfere with the blood-making or blood-regulating functions of the liver, and spleen, as the compounds of lead, copper, mercury, arsenic, and antimony, cause even in small doses the appearance of this substance in the urine; and when taken in doses sufficiently large to produce poisonous effects the quantity is greatly increased.

In malarial fever the constituent of the blood which suffers to the greatest and most essential degree is the colored blood corpuscle.

In yellow fever the constituent of the blood which suffers to the greatest and most essential degree is the albumen and its modification, fibrin.

The peculiar action of the poison in the former upon the colored blood corpuscles induces a distinct train of symptoms, and establishes distinct recognizable lesions, characterized chiefly by the deposit of pigment matter in certain organs; while in the latter the poison causes such changes in the albumen and fibrin as lead to the formation of non-nitrogenous and nitrogenous materials, some of which, as the oil and modified fibrin, are arrested or accumulated in certain organs, as the heart, liver and kidneys.

During the active stages of both yellow and malarial fever, phosphorus and the compounds of phosphorus in the nervous structures, as well as sulphur and the compounds of sulphur in the muscular structures, undergo more rapid changes than in the normal state; and phosphoric acid and the phosphates, and sulphuric acid and the sulphates, appear in increased quantities in the urine when the kidneys perform their offices. The waste of phosphorus and of its compounds in the nervous structures during the active stages of the disease is greater than the supply of these materials through the food. The nervous disturbances and debility characteristic of these fevers, as well as of others, are in a measure due to those rapid changes in the phosphorescent materials of the nervous structures, and especially of the central ganglionic cells.

In many cases of yellow fever, and in that form of paroxysmal fever called malarial hematuria, the function of the kidneys is impaired, and neither the urea nor the mineral acids are increased in the urine; while at the same time they accumulate in the blood, and exert deleterious effects upon the nervous system and blood. The increase of the urea and of phosphoric and sulphuric acids during the active stages of these diseases should not, therefore, be considered as anything peculiar and as at all distinguishing them from other fevers. It is only the tendency to congestion and alteration of the excretory structures of the kidneys that characterize yellow fever.

The peculiar intoxication and nervous symptoms, as well as the black vomit of yellow fever, are intimately associated with suppression of the urinary excretion. In many cases I have found the black vomit of yellow fever to give strong alkalin reaction from the presence of ammonia resulting from the urea eliminated by the gastric mucous membrane. I have also detected by repeated analysis, urea in large amount in the brain, heart, liver, spleen, muscles, and blood in yellow fever. In this disease, suppression of the action of the kidneys is more to be dreaded than black vomit, which it often precedes and induces.

The increase of these constituents of the urine is referable to the same cause—that is, increased chemic change—in both fevers, although it is evident that the nature of these chemic changes, and the special constituents involved may differ in each disease.

During the slow action of the malarial poison, as well as during the active stage of the paroxysm, important changes take place in the liver and spleen which are wholly different from the changes of these organs in yellow fever. In malarial fever, in both the liver and spleen, the colored blood corpuscles are destroyed in large numbers, and the coloring matter resulting from the disintegration of the colored corpuscles accumulates in them, and in conjunction with other changes in the nutritive processes of these organs produce those characteristic alterations of the normal color. In fatal cases, cellulose is found both in the liver and spleen, while grape-sugar is absent from the liver. The bile is also altered, both in chemic constitution and physical properties.

In yellow fever there is no destruction of colored blood corpuscles either in the spleen or liver, and no deposit of pigment matter, while *oil* is deposited in large amount in the liver, which together with the bile, impart to this organ a yellow color far different from the dark slate or bronze color of the malarial liver. The spleen is comparatively unaltered in yellow fever. Both cellulose and grape-sugar are found in the liver of yellow fever.

That the chemistry of the body is deranged in a definite manner in malarial fever is evident from the changes of the excretions. During the chill, and at the commencement of the hot stage, phosphoric acid disappears almost entirely from the urine. As the hot stage progresses, and the febrile action and the heat begin to decline, there is an augmentation of phosphoric acid. The uric acid is either increased or remains at the normal standard during the chill, disappears almost entirely during the fever, and then increases rapidly and rises to a high figure after the subsidence of the febrile excitement, and often continues for days, two, three, or even six times more abundant than in the normal state, as I have shown by a large number of observations published twelve years ago.

The sudden variations in the physical and nervous phenomena of malarial fever are accompanied by equally sudden and marked anatomic lesions and changes in the excretions. No such variations in the phosphoric or uric acids are observed in yellow fever. The poison inducing malarial fever acts in a definite manner, and is governed by definite affinities and relationships, and produces a type of disease distinct from yellow fever. The malaria of the swamps and marshes can only generate paroxysmal fever.

In the vast majority of cases of malarial fever, albumen does not appear in the urine. This constituent of the blood may, however, be present in the urine in malarial fever under certain circumstances.

1. Its presence in the urine of malarial fever may be due to preceding disease of the kidneys, of the liver, or heart.

2. To the prolonged action of the malarial poison, and the structural alterations induced by it in the spleen, liver, and kidneys.

3. To the congestion of the kidneys from cold, or

from the impaction in the capillaries of pigment matter, or from the irritant action of the malarial poison upon the excretory structures in cases which have suffered with repeated attacks of intermittents.

It is the exception to the rule to find albumen in the urine in malarial fever; it is the exception to the rule to find albumen absent from the urine of yellow fever.

Even in those cases where the prolonged action of the malaria has produced profound structural alterations of the liver, consisting in the extensive deposit of black pigment granules within and around the capillaries of the liver, obliteration of many of the branches of the portal system within the lobules, and in the hardening and contraction of the entire organ, albumen is rarely present in the urine. I have examined the urine carefully without detecting albumen in a number of cases of ascites and extreme dropsical infiltration of the lower extremities, produced by the hardening and contraction of the liver in chronic malarial poisoning. I have observed cases, however, in which the kidneys were structurally altered by the malarial poison in a manner somewhat similar to the liver, in which albumen was a constituent of the urine.

A certain proportion of such cases may be referred to the causes which ordinarily lead to structural alterations of the kidneys, as the excessive use or abuse of ardent spirits, and the effects of exposure to wet and cold and extreme temperature; but there are cases of albuminuria which can be explained only upon the supposition that they are due to the structural alterations of the kidneys induced by the prolonged action of the malarial poison. And this condition of the urine is not to be referred to the watery condition of the blood induced by the destruction of the colored corpuscles and diminution of the albumen and fibrin; for the state of extreme anemia frequently induced by the action of the malarial poison is never attended by albuminuria, unless there be some structural alteration of the kidneys.

In that form of malarial fever characterized by complete jaundice, intense vomiting and nausea, and hemorrhage from the kidneys, which has received different names at different times and in different countries, and which is no "new disease" even in these Southern States, the hemorrhage from the kidneys is preceded by congestion of these organs, and is attended with desquamation of the excretory cells, and tubuli uriniferi of these organs.

Malarial hematuria (hemorrhagic malarial fever—new disease—up-country yellow fever,) as a general rule, occurs only in those who have suffered from repeated attacks of intermittent fever, or who have been exhausted by a prolonged attack of remittent fever; and while some of the symptoms, as the nausea, incessant vomiting (and in extreme cases black vomit,) deep jaundice, and impaired capillary circulation, resemble those of yellow fever, yet there are marked differences, similar to those already indicated, as distinguishing malarial and yellow fever.

The presence of the albumen in the urine of this so-called "malarial hematuria" is attended also with the presence of colored blood corpuscles, excretory cells of the kidneys, and the tubuli uriniferi, impacted ofttimes with altered blood corpuscles. I have even detected the Malpighian corpuscles containing altered blood corpuscles, and deeply stained by the coloring matter of the blood. As a general rule in yellow fever, the tubuli uriniferi are loaded with yellow granular, albuminoid, and fibroid matter.

In those cases of malarial hematuria which have come under my observation there was evident congestion of the kidneys, attended with desquamation of the excretory cells and coats of the tubuli uriniferi and active hemorrhage. In some of these cases immense quantities of green biliary fluid were vomited, and the patients died in a state of apparent collapse. As a general rule, suppression of the functions of the kidney is a fatal sign, and, as in yellow fever, may be attended with convulsions, coma and delirium. A careful examination of the blood in malarial hematuria reveals great diminution of the colored corpuscles and fibrin.

The pathologic changes observed after death are characteristic of malarial fever: enlarged slate-and-bronze liver, with pigment granules; enlarged and softened spleen, filled with disorganized colored corpuscles and pigment granules; gall-bladder distended with thick, ropy bile, presenting when seen *en masse* a greenish-black color, and in thin layers a deep yellow. As much as one thousand grains of bile of high sp. gr. (1036) have been obtained from the gall-bladder, while in yellow fever not more than one hundred and twenty grains of bile are, as a general rule, contained in the relaxed gall-bladder.

I have thus clearly demonstrated that malarial hematuria is related to the various forms of true malarial fever (intermittent, remittent, and congestive,) and in fact is only one of the phases of this fever which may at any time be assumed after the alterations of the blood, liver, and spleen induced by the prolonged action of malaria. I have also clearly shown that it is distinct from yellow fever, although it may have some symptoms in common, as jaundice, black vomit, and albuminuria.

The treatment of malarial hematuria should be conducted upon the same general principles which should guide us in the treatment of pernicious intermittent, remittent, or malarial fever; with this addition, that attention should be paid to the condition of the kidneys, and they should be relieved by cut cups and counter-irritation. The bowels should be freely opened by a mercurial (calomel is the best preparation) combined with quinin. Ten grains of calomel and ten grains of quinin is a useful combination. Quinin should be freely given. The strength should be supported by nutritious diet (beef-tea and milk punch,) given by the rectum if the stomach will not bear it. Alcoholic stimulants should be used without any fear of injury. The action of the skin should be promoted by the hot-air bath and steam bath.

In brief, the *strength must be supported and the paroxysm arrested by quinin; the liver and bowels and portal system must be unloaded; the congestion of the kidneys must be relieved;* and during convalescence the blood must be enriched with pure and nutritious diet, and iron, and a gentle action of quinin maintained.

The following cases will serve to illustrate the effects of a preceding attack of malarial paroxysmal fever, upon the progress of the symptoms and pathology of yellow fever:

PATHOLOGY OF YELLOW FEVER RELATIONS TO MALARIAL FEVER.

Case 17.—Remittent fever followed by yellow fever; patient reduced in strength and anemic before the attack of yellow fever; fatal issue; liver presented the marks of both yellow fever and malarial fever. Mark Curtis; age 22; native of Germany. Entered Charity Hospital, Oct. 18, 1873. Patient states that on October 3 he was seized with vomiting followed by high fever; the vomiting and fever returned every alternate day, until his entrance into the hospital. Previous to this time had been exposed to the malaria of the swamps; came to New Orleans on October 3, 1873. Has resided in America five years, and in Louisiana since October 1 of the present year.

October 18. Patient very anemic and sallow; lips bloodless; gums pale; tongue very pale and flabby, with edges indented by the teeth; pulse 92; respiration 26; temperature of axilla 101.5 degrees.

October 19, 8 A.M. Patient complains of pain in upper and lower extremities. Tongue pale, flabby and furred at the center. Patient anemic, feeble, lethargic and without appetite. Urine abundant and without albumen. Bowels not constipated. Pulse 66; respiration 22; temperature 99.5 degrees. 7 P.M., pulse 72; respiration 20; temperature 100 degrees. Tongue pale, almost as colorless as his face, and furred in the center; gums pale and bloodless; conjunctiva a light yellow, not congested. Patient complains of dizziness in his head. Has been placed under the influence of quinin and has taken about 20 grains during the day.

October 20, 8 A.M. Complains of pain in head, back and lower extremities. Presents the appearance of great anemia about the lips and face, although the surface is now somewhat congested, and the patient has high fever. Pulse 104; respiration 22; temperature of axilla 104.5 degrees; 7 P.M. Patient complains of pains in all his bones. Urine contains no albumen. Pulse 112; respiration 26; temperature of axilla 105.4 degrees. Has taken 10 grains of sulphate of quinia at 10 A.M. and at 12 M. (20 grains in all) without any apparent effect upon the fever. Ordered 20 grains of sulph. carbolate of sodium, every three hours during the night, until 60 grains are taken. Ice, brandy and carbonic acid water and iced milk.

October 21, 8 A.M. Complains of pain in head, back and extremities; capillaries of eyes and surface congested; conjunctiva and surface generally present a yellow jaundiced hue. Urine contains no albumen, light colored and abundant. Pulse 80; respiration 16; temperature of axilla 100 degrees. As there appeared to be a remission in the fever, ordered 3 grains of sulphate of quinia, every three hours, until 24 grains are taken. Continue fragments of ice, carbonic acid water and brandy and iced milk. 7 P.M., the quinin has not prevented the rise of the fever. Pain in the head and bones continues. Pulse 90; respiration 22; temperature of axilla 103.7 degrees. Continue sulpho-carbolate of sodium, ice, brandy and carbonic acid water and iced milk.

October 22, 8 A.M. Patient restless; slept not at all during the night. Complains of pain in right side in region of liver. Capillaries of surface congested, but tongue and gums anemic, great prostration, patient desponding, depressed and restless. Pulse 96; respiration 22; temperature of axilla 103 degrees. Continue sulpho-carbolate of sodium. 7 P.M. Pains in bones and extremities continue. Pulse 86; respiration 24; temperature of axilla 104 degrees.

October 23, 8 A.M., capillary congestion increasing. Complains of pain in right side; loss of appetite, nausea and restlessness. Pulse 94; respiration 23; temperature 100.9 degrees. 7 P.M., pulse 80; respiration 26; temperature 104.5 degrees.

℞ Quinia sulph . . . . gr. xx.

Divide into four powders; one powder every three hours.

October 24, 8 A. M. Capillary circulation very imperfect. When fingers are pressed upon the purplish congested surface a white mark is left into which the blood slowly returns. Conjunctiva yellow and congested. Intense jaundice. Spirits greatly depressed; restless; complains of pain in right side. Pulse, 94; respiration, 21; temperature, 100.4.

℞ Sulpho-carbolate of sodium . . . . ʒi

Divide into three powders; one powder every three hours.

℞ Quiniae sulph.; pulv. rhei . . . . . āā ℈i

Mix; divide into two powders; one powder every two hours.

7 P. M., pulse 70; respiration, 20; temperature of axilla, 102. Breath very offensive. Body emits a foul odor.

October 25, 8 A. M. Bowels constipated, breath and odor of body very foul. Complains of pain in both sides and abdomen. Epigastrium and abdomen generally very tender on pressure. Tongue, gums and lips anemic. Surface of face, trunk and extremities purplish yellow. Conjunctiva congested and very yellow. Tongue white and furred at center; large with edges indented by the teeth; this condition of the tongue is attributed to the preceding action of malaria. Urine loaded with albumen and biliary matters. Pulse 78; respiration 22; temperature of axilla 101 degrees.

℞ Quinia sulph.; pulv. rhei, ... āā ℈i

Mix; divide into four powders. Take one powder every three hours.

Continue sulpho-carbolate of sodium with milk, brandy and ice as before.

At 7 P. M. the rhubarb has caused small action in the bowels, and the patient says that the pain in the side and bowels is much less and that he feels better. Pulse 70; respiration 19; temperature of axilla 101 degrees.

October 26. 8 A. M. Pulse 93; respiration 24; temperature of axilla 100.5 degrees. The quinin appears to have reduced the temperature, and the action on the bowels caused by the rhubarb appears to have afforded much relief. Continue sulpho-carbolate of sodium, 20 grains every four hours. 6 P. M., pulse 71; respiration 25; temperature of axilla 99.2 degrees. The patient vomited his food about 4 P. M. Continue brandy and iced milk.

October 27, 8 A. M. Pulse 72; respiration 20; temperature of the axilla 99.6 degrees. Great capillary congestion; deep jaundice, nausea and vomiting. Continue sulpho-carbolate of sodium. 7 P. M., pulse 62; respiration 18; temperature of axilla 99.2 degrees. Urine scanty and loaded with albumen casts, granular matter, excretory cells and biliary matter.

October 28. 8 A. M. Patient presents a fearful appearance; great capillary congestion, with yellow and purple surface. Yellow congested eyes, urinary excretion scant and loaded with albumen and casts. Has vomited curdled milk streaked with dark flakes. Pulse 60; respiration 16; temperature of axilla 97.2 degrees. The pulse, respiration and temperature give no indication of the extreme danger of this case. Continue sulpho-carbolate of sodium, 20 grains every three hours; ice water injection to rectum; iced milk; brandy and carbonic acid water; ice bag to epigastrium. Black vomit. Notwithstanding the occurrence of black vomit and the great capillary congestion of the surface, the tongue and gums still continue pale. 7 A. M., pulse 58; temperature 99.1 degrees. Vomiting of black vomit continues. The ice-cold enema appeared to restore the action of the kidneys temporarily. Continued to throw up black vomit during the night.

October 29. Died at 6:30 A.M.

The following table presents the relations of the pulse, respiration and temperature:

Date. 1893.	Pulse.		Respiration.		Temperature.	
	M.	E.	M.	E.	M.	E.
Oct. 18 . . .		92		26		101.5°
“ 19	66	72	22	20	99.5°	100.0
“ 20	104	112	22	26	104.5	105.4
“ 21	80	90	16	22	100.0	103.7
“ 22	96	86	22	24	103.0	104.0
“ 23	94	80	23	26	100.9	104 5
“ 24	94	70	21	20	100.4	102.0
“ 25	78	70	22	19	101.0	101.0
“ 26	93	71	24	25	100.5	99.2
“ 27	72	62	20	18	99.6	99.2
“ 28	60	58	16	20	97.2	99.1

Necropsy.—Three hours after death. Body still warm; weather cold and frosty; temperature of cavities of heart 97.0 degrees. There had evidently been no rise of temperature after death in this case. Exterior: Deep yellow color. Conjunctiva of eyes yellow. Dependent portions of body mottled. Surface presents the appearance of great capillary congestion. Lips and gums pale and of the same color as the skin of the face. Tongue also pale and bloodless. Throughout the illness of this patient the tongue has been very pale, and I called the attention of my medical students to the effects of the preceding malarial disease, in rendering the blood watery, and in affecting the color of the tongue. The appearance of the tongue in this case was decidedly different from that of uncomplicated yellow fever.

Thorax: Recent pleuritic adhesions in the lobes of the right lung; the patient during life had complained of pain in right side, as well as in abdomen. Dependent portions of both lungs much congested. We have in this pleuritic inflammation, proof that inflammation may arise, in a system depressed by the action of the poisons of malarial and yellow fever. Heart: Cavities distended with dark fluid blood. When abstracted, the blood from the heart formed a loose voluminous soft-coagulum which did not completely gather up the colored blood corpuscles. Reaction of blood neutral. Red blood corpuscles greatly diminished; colorless corpuscles very abundant. Under the microscope some of the colored corpuscles presented a crenate stellate appearance, but the vast majority were normal in appearance. After prolonged and careful microscopic examination with magnifying powers ranging from 420 to 1050 diameters, I was unable to discern any bacteria, or living animalculæ in the blood of the heart. Blood thin and watery. Serum golden colored. Muscular fibers of heart, pale and of a yellow color. Upon microscopic examination, the transverse striæ of the muscular fibrilla are to me indistinct, and granular matter and oil globules were numerous; but the amount of oil and granular matter appeared to be less than in uncomplicated cases of yellow fever. The patient was anemic and emaciated before the attack of yellow fever.

Abdominal cavity: Liver. This organ presented on the exterior a bronzed and mottled color, resembling a liver of malarial fever, which was in the early stages of fatty degeneration. The general appearance of this liver would lead the superficial observer to repeat the assertion which unfortunately has been too often made by those who have enjoyed every opportunity to crush the error, "*that there is no characteristic lesion of the liver in yellow fever, and that this disease has no pathology.*" The surface of the liver presented a mottled color of bronze and deep yellow. Many of the lobuli presented a deep yellow color, like the liver of uncomplicated yellow fever. When sections of the liver were carefully washed, the distinct deposit of yellow fever was everywhere discernible and the more deeply colored portions were chiefly in the peripheral capillary network of each lobule.

Under the microscope the liver was found to be loaded with oil globules and yellow granular matter, and also with numerous masses of hematin, deposited chiefly in the peripheral system of portal capillaries. The peculiar color of the liver, therefore was clearly referable to the combined action of yellow fever and malarial fever. The presence of the pigment particles revealed the previous action of the malarial poison. Gall bladder flaccid; contained no bile, and only a small quantity, about twenty drops of a thick cream-like semi-liquid substance; which also filled and obstructed the gall ducts under the microscope. This was found to consist chiefly of desquamated epithelium from the mucous membrane of the gall bladder. Mucous membrane of gall bladder, thickened and intensely congested, presenting a deep red and purple color. Spleen enlarged and softer than in uncomplicated yellow fever. This organ contained much pigmentary matter.

Kidneys: Congested. When sections were made and washed, the kidney structure presented the characteristic yellow color of yellow fever. Sections with Valentine's double-bladed knife, under the microscope, presented the usual appearance of Malpighian corpuscles and tubuli uriniferi impacted with oil globules, granular matters and detached cells.

The urinary bladder: Contained about one fluid drachm of turbid yellow urine, loaded with colorless corpuscles, casts of the tubuli uriniferi, detached excretory cells from tubuli and pavement epithelium from pelvis of kidney, ureter and urinary bladder.

Spermatozoa: The urine contained albumen. I have observed spermatozoa in the urine of several cases of yellow fever after death.

Stomach and intestinal canal: The stomach contained about one pint of a mixture composed of milk and black vomit. A portion of this milk soon coagulated. Under the microscope the contents of the stomach consisted of the oil globules of the milk, masses of hematin, detached cells from the mucous membrane of the stomach and also the spirules and matters of a delicate fungus resembling the torula, which is very common in the black vomit of yellow fever. I have frequently observed the same fungus in the urine of yellow fever, after it has been allowed to stand for a certain time. The sulpho-carbolate of sodium did not prevent the formation of this fungus. The mucous membrane of the stomach congested and ecchymose. The mucous membrane of the ileum and colon greatly congested, presenting a brilliant injected appearance. In portions the mucous membrane was softened, eroded and covered with effusive blood.

The peritoneum exhibited evidences of recent inflammation. The omentum was intensely injected. Coagulable lymph of a yellow color and in some places of a red color was thrown out in small amounts upon the surface of the peritoneum. The inflammation of the pleura and peritoneum in this case may have been excited by the cold weather and the sudden changes of the temperature of the ward, in which the stoves had not been erected.

In the preceding case, we have a complication of diseases, namely, malarial fever, yellow fever, pleuritis and peritonitis.

It is a matter of great moment to determine whether the malarial poison under any circumstances induces fatty degeneration of the heart, liver and kidneys. If the malarial poison acted in a manner similar to that of yellow fever in producing fatty degeneration of these organs, a strong argument could be established for the identity of these two diseases in their origin and manifestations. I have endeavored to investigate this question thoroughly, by carefully observing the individual cases and noting the chemic and microscopic changes after death, in the blood and different organs, and especially in the liver, spleen, kidneys and heart. In no instance have I observed the production of fatty degeneration of the heart, liver and kidneys in malarial fever. All the various forms of malarial fever have been subjected to critical investigation, including remittent fever, pernicious, malarial or congestive fever, malarial hematuria, quotidian, tertian and quartan intermittents and chronic malarial poisoning.

If in any condition, fatty degeneration of the heart, liver and kidneys results from the action of the malarial poison, it should be most clearly shown in prolonged cases of malarial poison. I selected the following from a large number of similar cases:

Case 1.—Chronic malarial poisoning, anemia, destruction of colored blood corpuscles, high temperature, fatal issue. Samuel Erhart; age 35; native of Germany; entered ward 13 Nov. 22, 1873, in a feeble depressed condition, with continued fever and sallow greenish-yellow complexion. Has been in America about eight months. In Europe his profession was that of an architect, but during his residence in America he has worked as a laborer in the low swamp lands of Louisiana. About eight weeks before entrance into the Charity Hospital, had a chill followed by fever, which has remitted occasionally, but has never been entirely absent. During his sickness has had scant supplies of food, and has had little or no medical attendance. The effects of the action of the malarial poison are manifest in his pale, sallow, greenish-yellow surface, pale lips and gums, muscular and nervous prostration, continuous febrile excitement, bloated countenance and swollen extremities. His present condition Nov. 22, 1873, is clearly referable to the action of the paludal poison of the swamps, bad diet and neglect.

November 23, patient pale, anemic, rapid pulse, high temperature. Nausea and vomiting of bilious matter. Quinin is retained with difficulty on the stomach and the patient takes little or no nourishment. November 24, A.M., temperature of axilla 105 degrees; pulse 112; respiration 21. Urine high-colored, but free from colored blood corpuscles and albumen. Chlorids greatly diminished. Nausea and jaundice. Great nervous and muscular prostration. 8 P.M., temperature of axilla 104.7 degrees. November 25, A.M., temperature of axilla 103 degrees, pulse 116. 8 P.M., temperature 104 degrees; delirious. Strength failing. November 26, A.M., temperature 102 degrees; P.M., temperature 103 degrees. Notwithstanding the great prostration of the patient, the fever still continues. We observe in the record of temperature no decided decline of the fever, but only fluctuations which correspond with those of the so-called bilious remittent fever. The disease has evidently run into this continued stage from the absence of all remedial agencies. It was impossible to produce any impression upon this patient by means of quinin, administered either by the mouth or rectum. November 27, patient died at 6 A.M.

Post-mortem, three hours after death. Body warm, limbs flaccid. Exterior, of dingy greenish-yellow color. Lower extremities edematous.

Thorax: The pericardium contained about two fluid ounces of golden yellow serum. Heart presented a deep color and firm texture. Under the microscope, no textural changes were observed in the muscular fibrillæ of the heart, and the microscopic characters, as well as the chemic constituents, were wholly different from those of yellow fever. Examination of the blood from the cavities of the heart: Light yellow fibrinous concretions were observed in both cavities. The fluid blood from the cavities of the heart, when viewed by the microscope was found to contain a large number of dark masses of hematin of various sizes, from one three thousandth of an inch to one seventy-five hundredth of an inch in diameter. These masses were similar in all respects to those observed in the spleen and liver. Specific gravity of blood from cavities of the heart, 1021; solid residue in 1,000 parts of blood, 73.00. Specific gravity of serum of blood, 1015; solid residue in 1,000 parts of serum, 58.00. The fluid blood from the cavities of the heart coagulated after abstraction, and numerous oil globules were observed upon the surface of the coagulum. Coagulum large and soft; fibrin diminished in amount. Lungs normal; hypostatic congestion of dependent portions of lungs. Stomach distended with gas; mucous membrane pale and anemic. Stomach contained partially digested food discolored by bile. Liver enlarged and hardened; deep slate color on the exterior and bronze within, and contained numerous black pigmentary particles, deposited chiefly in the peripheral network of the portal capillaries. Spleen enlarged and softened and loaded with altered blood corpuscles and dark masses of hematin. Gall bladder flaccid and contained about fifty grains of thick mucus, which was loaded with the desquamated cells of the mucous membrane of the gall bladder. Kidneys normal, with the exception of slate-colored spots, about one and one-half inches in diameter, on the surface of each kidney; and when thin sections of the discolored portions were examined under the microscope. the discoloration was found to be due to the deposit of black pigmentary particles in the capillaries.

Chapter III.

CHANGES OF THE TEMPERATURE, PULSE AND RESPIRATION IN YELLOW FEVER.

Owing perhaps chiefly to the sudden origin, rapid progress, and singularly fatal nature of yellowfever, as well as the infrequency of the use of the thermometer in the investigation of the disease, but few facts of value relating to the definite degrees of temperature in the different stages of the disease can be found in the writings of various observers. Almost all observers, however, as Benjamin Rush,[15] J. Deveze,[16] Wm. Currie,[17] C. Caldwell,[18] Samuel Jackson,[19] P. Chas. A. Louis,[20] W. Arnold,[21] R. Jackson,[22] Lionel Chalmers,[23] John Lining,[24] A. M. F. Savarésy,[25] William T. Wragg, Daniel Blair,[26] John Davy,[27] Schmidtlein,[28] and many others, concur in the statement that in the early stage of reaction or febrile excitement the temperature is elevated to a greater or less degree, in different cases, above the standard of health; and still further, that this elevation of temperature is not permanent, but at the end of from two to five days, in most cases, is succeeded by a decided fall; and although the symptoms may be of the gravest character and the patient *in extremis*, neither the elevation of the temperature nor the frequency of the pulse give any true indication of the danger.

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[15] Medical Inquiries and Observations (4 vols. octavo), Philadelphia, 1809.

[16] Recherches et Observations sur la Maladie Epidemique qui à régne à Philadelphia, en 1793; Dissertation sur le Fievre Jaune, Traité de la Fievre Jaune, etc.

[17] Treatise on the Synochus Icterodes, etc., 1794.

[18] Facts and Observations relative to Yellow Fever.

[19] An Account of the Yellow or Malignant Fever which appeared in the city of Philadelphia in the Summer and Autumn of 1820.

[20] Anatomic, Pathologic, and Therapeutic Researches in Yellow Fever of Gibralter, 1828; translation, Boston, 1839.

[21] A Practical Treatise on the Bilious Remittent Fever (Yellow Fever) etc., London, 1840.

[22] Treatise on the Fevers of Jamaica. London, 1791.

[23] Accounts of the Weather and Diseases of South Carolina. London, 1768.

[24] A Description of the American Yellow Fever, in a letter to Dr. Whytt.

[25] De la Fievre Jaune, etc. Naples, 1809.

[26] Some Account of the last Yellow Fever Epidemic of British Guiana. Third edition, London, 1852.

[27] Notes to Blair's Account of the last Yellow Fever Epidemic of British Guiana.

[28] On the Temperature in Diseases, by Dr. C. A. Wunderlich. (Sydenham Society, page 405.)

Dr. John Lining, of Charlestown, S. C., appears to have been the first physician who recorded definite observations on the variations of the temperature in yellow fever. In his description of the American yellow fever, contained in a letter addressed to Dr. Robert Whyte, Professor of Medicine in the University of Edinburgh, dated Charlestown, Dec. 14, 1753, and published March 7, 1754, the following general observations relate to the changes of the pulse, respiration and temperature in this disease: "After a chilliness and horror with which the disease generally begins, a fever succeeded, in which the pulse was very frequent till near the termination of the fever, and was generally full, hard, and consequently strong; in some, it was small and hard, in others, soft and small; but in all these cases, it frequently varied in its fullness and hardness. Toward the termination of the fever, the pulse became smaller, harder and less frequent. In some, there was a remarkable throbbing in the hypochondria; in the latter of which it was sometimes so great that it caused a constant tremulous motion of the abdomen.

"The heat generally did not exceed 102 degrees Fah.; in some it was less; it varied frequently, and was commonly nearly equal in all parts, the heat about the precordia being seldom more intense than in the extremities, when they were kept covered. In the first day of the disease, some had frequent returns of a sense of chilliness, though there was not any abatement of their heat. In a few, there happened so great a remission of the heat for some hours, when at the same time the pulse was soft and less frequent, and the skin moist, that one from these circumstances might reasonably have hoped that the fever would only prove a remittent or intermittent. About the end of the second day, the heat began to abate. . . .

"The respiration was by no means frequent or difficult, but was soon accelerated by motion, or the fatigue of drinking a cup of any liquid. . . .

"A nausea, vomiting or frequent retching to vomit, especially after the exhibition of either medicines or food, came on generally the third day, as the fever began to lessen; or rather as the fullness of the pulse, heat and disposition to sweat began to abate. Some, indeed, but very few, on the first day had a vomiting either bilious or phlegmatic. . . . .

"The strength was greatly prostrated from the first attack. The pain in the head, loins, etc., of which they had complained before the attack, was greatly increased, and in some the pain in the forehead was very acute and darting; but these pains went off generally the second day. The face was flushed, and the eyes were hot, inflamed, and unable to bear much light. . . .

"The fever accompanied with these symptoms terminated on the third day, or generally in less than seventy-two hours from the first attack, not by any assimilation, or concoction and excretion of the morbid matter; for if by the latter, there would have been some critical discharge by sweat, urine, stool or otherwise, none of which happened; and if, by the former, nothing then would have remained but great debility. No; this fever did not terminate in either of these salutary ways, excepting in some, who were happy enough to have the disease conquered in the beginning by proper evacuations, and by keeping up a plentiful sweat, till the total solution of the fever by proper mild diaphoretics and diluents. But those who have not that good fortune, however tranquil things might appear at this period, (as great debility and a little yellowness in the whites of the eyes seemed to be the chief complaints, excepting when the vomiting continued), yet the face of affairs was soon changed; for this period was soon succeeded by the second *stadium*,—a state, though without any fever, much more terrible than the first; the symptoms in which were the following:

"The pulse immediately after the recess of the fever, was very little more frequent than in health, but hard and small. However, though it continued small, it becomes soon afterward slower and very soft; and this softness of the pulse remained as long as the pulse could be felt. In many, in this stage of the disease, the pulse gradually subsided, until it became scarce perceptible; and this notwithstanding, all the means used to support and fill it; and when this was the case, the icteritious-like suffusion, the vomiting, delirium, restlessness, etc., increased to a great degree. In some, the pulse often being exceedingly small and scarce perceptible, recovered considerably its fullness; but that favorable appearance was generally of but short continuance.

"The heat did not exceed the natural animal heat; and when the pulse subsided, the skin became cold and the face, breast and extremities acquired somewhat of a livid color. . . . The respiration was natural or rather slow. . . . The vomiting or retching to vomit increased, and in some was so constant that neither medicines nor ailments of any kind were retained. Some vomited blood; others only what was last exhibited, mixed with phlegm; and others again have what is called the black vomit. The retching to vomit continued a longer or shorter time according to the state of the pulse; for as that became fuller, and the heat greater, the retching to vomit abated, and *é contra*. . . .

"The debility was so great that if the patient was raised erect in the bed, or in some, if the head was only raised from the pillow, while a cup of drink was given, the pulse sank immediately and became sometimes so small that it could scarcely be felt; at this time they become cold, as in a horripilation, but without the anemic-like skin; their skin became clammy, the delirium increased, their lips and skin especially about the neck, face and extremities, together with their nails, acquired a livid color. . . .

"There were many small spots, not raised above the skin, which appeared very thick on the breast and neck; but less so on the extremities, and were of a scarlet, purple or livid color. In women the menstrua flowed, and sometimes excessively, though not at their regular periods.

"There was such a putrid dissolution of the blood in this *stadium* of the disease, that, besides the vomiting of blood formerly mentioned, and the bloody urine soon to be taken notice of, there were hemorrhages from the nose, mouth, ears, eyes, and from the parts blistered with cantharides. Nay, in the year 1739, or 1745, there were one or two instances of a hemorrhage from the skin, without any apparent puncture or loss of any part of the scarf-skin.

When this *stadium* of the disease terminated in health, it was by a recess or abatement of the vomitings, hemorrhages, delirium, inquietude, jactatations, and icteritious-like suffusion of the skin, and white of the eyes; while at the same time, the pulse became fuller and the patient gained strength, which after this disease was very slowly. But when it terminated in death, these symptoms not only continued, but sooner or later increased in violence, and were succeeded with the following, which may be termed the third *stadium* of the disease, which quickly ended in death.

"The pulse though soft became exceedingly small and unequal; the extremities grew cold, clammy, and livid; the face and lips in some were flushed; in others, they were of a livid color; the livid specks increased so fast that in some the whole breast and neck appeared livid; the heart palpitated strongly; the heat about the precordia increased most; the respiration became difficult, with frequent sighing; the patient now became anxious and extremely restless;

the sweat flowed from the face, neck and breast; blood flowed from the mouth or nose or ears, and in some, from all these parts at once; the deglutition became difficult; the hiccoughs and subsultus of the tendons came on and were frequent; the patients trifled with their fingers, and picked the nap of the bedclothes; they grew comatose, or were constantly delirious. In this terrible state, some continued eight, ten or twelve hours before they died, even after they had been so long speechless, and without any perceptible pulsation of the arteries in the wrists; whereas, in all other acute diseases, after the pulse in the wrist ceases, death follows immediately. When the disease was very acute, violent convulsions seized the unhappy patient, and quickly brought this *stadium* to its fatal end. After death, the livid blotches increased fast, especially about the face, breast and neck, and the putrefaction began early, or rather increased very quickly.

"This was the progress of this terrible disease through its several *stadia.* But in hot weather, and when the symptoms in the first stage were very violent, it passed through these stages, as Dr. Warren has likewise observed, with such precipitation that there was but little opportunity of distinguishing its different *stadia;* the whole tragedy having been finished in less than forty-eight hours."

Dr. Lining also records observations on the changes of the tongue, skin and urine which have been omitted from the preceding quotation, in order to present in a connected manner his accurate accounts of the changes of the pulse, capillary circulation, blood, respiration and temperature.

In his prognostics, Dr. Lining held that a sediment in the urine in the first and second day of the disease is bad, and the more copious the sediment is, the greater is the danger; bloody urine and all hemorrhages, excepting slight ones from the nose are bad; and the more copious they are, the greater is the danger; a suppression of urine, especially in those who in the course of the disease have had large discharges that way, is a certain sign of the quick approach of death.

Dr. Lining held that the yellow fever of America was a pestilential, specific contagious fever, imported from the West Indies, "which continues two or three days and terminates without any critical discharge by sweat, urine, stool, etc., leaving the patient excessively weak, with a small pulse, easily depressible by very little motion, or by the erect posture; and which is soon succeeded with an icteritous color in the white of the eyes and the skin, vomiting, hemorrhages, etc., and these without being accompanied with any degree of a febrile pulse and heat."

Subsequent observations have tended to confirm the accuracy of Dr. John Lining's comprehensive description; and the voluminous writings of Dr. Benjamin Rush, of Philadelphia, Drs. Samuel Latham Mitchell, and Edward Miller, of New York, and others a century later added literally nothing to the fullness of his description; but on the contrary these writers, and especially Benjamin Rush, caused much subsequent confusion by confounding the endemic malarial, paroxysmal fevers of the paludal regions of North America with the specific yellow fever of the West Indies.

Savarésy[29] who observed yellow fever in Martinique in 1803 and 1804, affirmed that the heat rises often as high as 38 or 40 degrees of Reaumur—equal to 117.00 or 102.2 Fahrenheit.

Dr. John Davy[30] published in 1814 the first of his remarkable papers containing the results of his experiments in animal heat; and in 1816 he began his observations upon the temperature of man in various climates, in various diseases and under various states of rest and exercise, fasting and repletion in various races;[31] and in 1828 he established the singular fact that the human body may manifest a high degree of heat after death.

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[29] De la Fievre Jaune en générale, et particulierement de celle qui a régne a la Martinique en l' an xi et xii 1803, 1804, 8vo. Naples, 1809.

[30] Philosophical Transactions of the Royal Society of London 1814, p. 590. Researches, Physiological and Anatomical, by John Davy, M.D. and R.S. Assistant Inspector of Army Hospitals, 1839. vi, pp. 141 248.

The following are the cases as reported by Dr. John Davy:

Case 1.—Aged 23 years; was admitted into hospital on July 30, 1828, laboring under symptoms (as it was supposed) of acute rheumatism, having some pain first in one shoulder, then in the other, followed by pain in the hips, attended with much pyrexia, and a very rapid pulse. He died on August 6, a few minutes after 7 A.M. As the weather at the time was very hot (Military Hospital in Valetta, Island of Malta), and dead animal matter putrefied rapidly, it was necessary to inspect the body as soon as possible. Accordingly, it was examined three hours and a half after death; when the temperature of the air of the room was 86 degrees. The appearances most remarkable, discovered on dissection, were extensive collections of matter in the right shoulder, among the muscles on each side of the spine of the scapula, with sinuses extending to the axilla, and around the capsule of the shoulder joint; and a lesion of the same kind, and as extensive, in the left hip close to the head of the femur, spreading through the glutei muscles; and marks of incipient inflammation (as ecchymosis) on the right hip. The viscera were apparently sound. The right cavities of the heart and the great vessels were distended with liquid blood. The body was slender but not emaciated. Its surface was warm; the deeply seated parts felt very hot, imparting a disagreeable sensation, almost like that of burning, to the hand in contact with them. The thorax was first opened and afterwards the abdomen. After partial exposure of the surface of the contents of these cavities to the air for about ten minutes, a thermometer was procured. Placed under the left ventricle of the heart it rose to 113 degrees, and under the liver, in contact with the lobulus spigelii, to 112 degrees.

Case 2.—Aged 27 years, a stout, robust man, died suddenly in barracks, on August 6, at about 1:30 A.M. The body was examined at 5 P.M. There was a good deal of reddish fluid in the ventricles, and at the base of the brain. The lungs were distended with black liquid blood, some of which had passed into the bronchii. There was very little blood in the cavities of the heart. The temperature of the air of the room was 86 degrees. As soon as the cavities of the thorax and abdomen were opened, the bulb of a thermometer was in succession placed under the left ventricle of the heart, and under the lobulus spigelii of the liver; in the former situation it rose to 108 degrees; in the latter to 107 degrees. Almost a quarter of an hour afterwards, introduced into the substance of the right lung, gorged with extravasated blood, it was 105 degrees.

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[31] Third Ed. Philadelphia, 1826, Res., Phys. and Anat., vi., p. 161.

The question will naturally be asked, Was the extraordinary temperature, in one case of 113 degrees, and in the other of 108 degrees, generated during life or after death? Dr. Varez had little hesitation in coming to the conclusion that it was generated before death, and generated probably in the same way as the ordinary degree of animal heat experienced in health, or the extraordinary degree witnessed in febrile diseases. *A priori*, the effort of the heat-generating process, whatever it may be, can hardly be limited. In many birds, it raises the temperature of the body to 109 degrees, when in perfect health, and in man to 101 degrees, at least in the tropics, without deranging health; and it is easy to conceive that by increased activity or energy, it may exalt the temperature to the common febrile height, or to a height greatly exceeding that. But, destitute of life, in the opinion of Dr. Varez, there does not appear to be in the body any source of heat, any power of generating it, that is known. Putrefaction had not taken place in these bodies; even if it had, and had made progress, and were it even at its greatest height of activity, it is doubtful if it would be equal to the production of the effect in question.

It is a matter of regret that the temperature of the preceding cases was not ascertained by Dr. Davy before as well as after death; it is only known that the skin of the first case the day before he died was pungently hot.

These observations by Dr. Davy were of great value, in that they should have put medical inquirers on their guard as to the extreme limits of the degree of animal temperature, and especially of the blood in deep-seated parts. If the temperature of the body be 113 degrees, three hours after death, and if generated during life, before death it must have been still higher. Similar investigations were instituted by Dr. Bennet Dowler fifteen years later, in 1843, 1844, 1858, on the *post-mortem* heat of yellow fever and other diseases, the results of which will be fully examined in a subsequent part of the present discussion.

Subsequently, Dr. John Davy[32] made observations on the pulse and temperature in yellow fever, during his service in the West Indies. Thus in his notes to the work of Dr. Daniel Blair, on the yellow fever which prevailed in British Guiana from 1837 to 1842, Dr. Davy states that, "unusual slowness of pulse was very often observed in the yellow fever of Barbadoes, and commonly accompanied with undue coolness of skin, especially in the extremities; it was a very remarkable feature of the disease after its first invasion—the time varying in different cases. . . . . From the few observations which were made with the thermometer, on the temperature of men laboring under yellow fever in Barbadoes during the last epidemic, it did not appear to be high—when highest not exceeding 104 degrees Fah. in the axilla. In two instances trial was made of the temperature after death (four hours); on one (Fitzpatrick), that of the brain was found to be 98 degrees, of the lungs 100 degrees, of the heart 101 degrees (left ventricle), 100 degrees (right ventricle); in the other (Reynolds), that of the brain was 98 degrees, that of the lungs 102 degrees, of the heart 104 degrees (left ventricle); 103 degrees (right); of the liver 103 degrees. For the latter observations Dr. Davy was indebted to his friend, Staff-Surgeon Dr. Spence, who in the capacity of principal medical officer of the garrison of St. Ann, had ample opportunities to study the disease, of which, until attacked by it, he zealously availed himself.

Dr. Arnold,[33] in his "Practical Treatise on the Bilious Remittent Fever" (yellow fever), devotes an entire section of his work (occupying 66 pages out of 320), to the illustration by tables and cases of the temperature of the system in the febrile diseases of Jamaica, and states in his preface that he considers the temperature of the system in febrile diseases to be one of the most formidable symptoms; and expressed the belief that the system when laboring under bilious remittent fever (yellow fever), "must inevitably give way to the cause which produces even a few degrees of thermal exaltation beyond 105 degrees of Fahrenheit's scale, and that all our remedies will have little effect if we find it 110 degrees during the first or second stages."

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[32] Some Account of the last Yellow Fever Epidemic of British Guiana. By Daniel Blair, M.D., Surgeon-General of British Guiana. Edited by John Davy, M.D., F.R.S., etc. London, 1852, pp. 77, 78.

The observations of Dr. Arnold were begun in 1819, and continued for twenty-five years, and he prefaces his observations on the changes of temperature in yellow fever, remittent fever, intermittent fever and typhoid fever, by the results of his investigations on the normal temperature of 107 subjects of all denominations, ages and sexes, including Creoles, black laborers, domestics, carpenters, mulattoes, white natives and Europeans; and the mean of that whole series was 98.19 degrees F., which Dr. Arnold considered a correct estimate of the temperature of the system in health in a climate like that of Jamaica. Dr. Arnold says: "*I believe there is no study more calculated to improve the healing art, or to throw more light on the nature of febrile diseases, than a minute attention to the state of the temperature of the system.*" . .

I shall next point out the deviations which occur in consequence of febrile or preternatural heat, by a series of cases and observations, for the purposes contemplated in the inquiry, and I think it will be seen that the system is not capable of having any great augmentation of heat from morbid causes, without incurring serious, and sometimes irreparable mischief. In fever, particularly of the remittent

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[33] A Practical Treatise on the Bilious Remittent Fever; its Causes and Effects. With Illustrative Tables and Cases on the Temperature of the System in the Febrile Diseases of Jamaica, etc. By W. Arnold, M.D., etc. London, 1840.

form, the whole power of the animal economy is deranged—all its healthy functions are suspended; digestion is only imperfectly performed; the mental energy is much weakened by the agent which causes the fever, the ardent heat and rapid circulation; the heart is laboring and appealing for relief; the whole system is screwed up, as it were, to its highest pitch of excitement. *The heat, at this time, is at its utmost limit, and if continued for many hours, the extinction of life* would in all probability result, in consequence of various changes or decompositions taking place in the blood. The exact degree of heat which is necessary to achieve this end has hitherto not been very accurately ascertained; it will require much diligent investigation to settle the point. The ingenious Dr. Hales believed "the heat of the blood in high fever to be 136 degrees." This is irreconcilable to anything I have witnessed during twenty-five years' practice. Boerhaave also made various experiments, and he seems to think that "*terribly mischievous work might be effected by a degree of heat above 106 degrees, for, as he asserts, the serum of the blood would be coagulated.*" And he further asserts *that the natural heat of the blood of a human creature approaches very nearly the degree of coagulation.* Hippocrates, 4 de Morb., 23, observes: "Such a degree of heat, if neglected or wrongly managed, may indeed dissipate the more thin and watery parts, and so gradually thicken the whole mass of blood; have bad enough effects that way, if it do not bring on a putredenous thinness." This was evidently but supposition; the serum of the blood or the white of an egg, requires a degree of heat to coagulate, which no living animal can bear, notwithstanding the puzzling tricks of fire-eaters and such mountebank impostors, who thrust themselves into ovens heated sufficient to bake a leg of mutton. Water at 108 degrees is too hot for the hands for any length of time; yet there are some persons, from custom, who can handle very hot pieces of iron; but such instances are only sufficient to form an exception to the rule and require no further comment."

Dr. Arnold records several fatal cases in which the temperature ranged from 108 to 110 degrees in the axilla. In three cases of yellow fever recorded by this physician, the temperature reached 110 degrees, and a similar degree of heat was observed in several cases during life by Dr. Bennet Dowler in 1843, 1844 and subsequently. In 1843 he began a remarkable series of investigations on the temperature of the heart and axilla during the various stages of yellow fever, and of the different organs after death. His original paper was first transmitted to, but not published in full in the *Western Journal of Medicine and Surgery* during the winter of 1843–44. Dr. Dowler continued his observations on the temperature in yellow fever and other diseases during a period of sixteen years,[34] and he established the fact that the temperature of the internal organs may attain after death a degree of heat equal to that witnessed by Dr. John Davy in the Island of Malta in 1828, viz., 113 degrees. Dr. Dowler confirmed the accuracy of the statement of Dr. Arnold, of Jamaica, that the temperature of the axilla in yellow fever may reach 110 degrees F.; and he also demonstrated that patients may recover from yellow fever whose temperature in the axilla has reached 109 degrees.

Dr. Dowler also demonstrated that, as a general rule, the temperature in the early stages of yellow fever is higher in fatal cases than in those which recover. Thus, in one series of cases of yellow fever, taken without selection, the following results were obtained: Fifteen patients who recovered, whose temperatures were taken at a period which averaged fifteen and one-third hours after the invasion, and afforded a mean temperature for the hand of 101.8 degrees, and for the arm-pit 104.84 degrees; nine persons who died, gave an average of, twenty-two and one-third hours after the invasion, for the hand, 103.62; for the arm-pit, 105.44 degrees.

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[34] Experimental Researches into Animal Heat in the Living and in the Dead Body, by Bennet Dowler, M.D., of New Orleans. New Orleans Medical and Surgical Journal, Vol. xii, 1856, pp. 54. 205. 289, 433. 603, 759, 433, 470; Vol. xvii, 1860, pp. 199, 356

The maximum of these classes coincided, but not the minimum. In those who died the average was higher; in the hand, the maximum reached by both was 107 degrees; the minimum in the hand among those who recovered was 95 degrees, and of those who died 99 degrees; the maximum of the former, in the axilla, 107 degrees; of the latter, 109 degrees; the minimum of the former, in the same region, 102 degrees, and of the latter 100 degrees, the latter being in *articulo mortis*.

Full justice will be done to the labors of Dr. Bennet Dowler in that portion of this chapter which relates to *post-mortem* heat in yellow fever.

Dr. Daniel Blair,[35] in his last published report on the "First Eighteen Months of the Fourth Yellow Fever Epidemic of British Guiana," says: "High temperature of the body seems to have persisted longer through the stages of the disease in this epidemic than in the past. There is great irregularity in the temperature of the surface; sometimes the forehead is the hottest part of the body, and occasionally the chest. The uncovered portions of the body in the latter stages are easily reduced in temperature; and thus while the exposed chest and extremities may feel cool to the touch, the axilla may raise the thermometer to 102 or 103 degrees. The highest temperature I have observed during the course of the disease was 107 degrees Fah."

According to Dr. Wragg,[36] in some cases of yellow fever, the thermometer indicated a temperature of 108 degrees in the arm-pit, and 105 degrees on the chest.

Dr. Robert N. Lyons,[37] has recorded valuable observations on the relations of the pulse and temperature in the yellow fever which prevailed in Lisbon, Portugal, in 1857. Dr. Lyons arranges the cases under five groups, viz.: 1, the algid form; 2, the sthenic form; 3, the hemorrhagic form; 4, the purpuric form; 5, the typhus form.

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[35] British and Foreign Medico-Chirurgical Review, April, 1856, Appendix, p. 6.

[36] Charleston Medical Journal and Review, 1855, Vol. x, p. 73.

[37] A treatise on fever or selections from a course of lectures on fever, being part of a course of theory and practice of medicine, delivered by Robert V. Lyons, K.C.C., etc. Am. Ed., 1861.

Algid Form:—"As a general rule, the vascular action was much below par in the algid form. I have observed cases in which the radial pulse was imperceptible for days; more commonly, however, the pulse at the worst, though excessively feeble, was not altogether obstructed, and by a delicate and practiced finger it could be felt and counted. Its rate was variable, more commonly below than above 100 per minute. I have noted it at 80, very fine, thready, weak and readily extinguishable, and at almost all intermediate rates to 120, with much the same character. The cardiac action was very feeble. I have not observed any diminution in the relative proportions of the first and second sounds of the heart; they were often diminished in tone and force, but preserved their relative characters of duration and intensity. I have not observed any want of accordance between the cardiac action and that of the arteries in the algid form, such as I shall have to speak of subsequently when treating of some of the other forms. In general, in this form the radial pulse could be taken as a measure of the cardiac action. When the pulse was feeble, the heart was likewise proportionately so. There was no exception to this rule, as my experience of the cases goes (p. 260).

"Sthenic Form:—This class of cases generally exhibits from the outset a remarkable elevation of temperature. It is common to find an increase of 3 degrees, 4 degrees or even 5 degrees Fah., and in some instances an increase of nearly 7 degrees has been observed. We have noted the thermometer in the axilla at 102 degrees, 103 degrees, 104 degrees, and in some instances it nearly touched 105 degrees Fah. This limit I have not actually seen reached in any case, though the mercury in some instances rose considerably above 104 degrees. (The instrument employed, an English one of the kind commonly used for hospital purposes, did not admit of more minute or accurate reading than by whole degrees (p. 266.)

"Rate of the Pulse in the Sthenic Cases:—The pulse, though often full and expanded, and occasionally hard and thrilling in this form, commonly ranges but little above 100 or 110 beats per minute. I have noted it in some cases at 112, 114, and in very rare instances so high as 120. In general, when the pulse runs above 115, the case passes into a low and typhoid state; on the other hand, I have seen well-marked pyrexia with a pulse at 60. It has fallen to 100, 90, 80, and even 70, and in two instances to 60, without very well marked diminution of the other pyrexial symptoms; in these latter instances it has always retained its force and expansion. Thus we not unfrequently have the combination of a pulse at 70, 80 or 90, or in general terms, under 100, while the thermometer in the axilla shows a persistent temperature above 100 degrees Fah.

"The following combinations of pulse rate and temperature were observed in the course of my investigations into the Lisbon epidemic; they are selected to show the absence of harmony and the frequent contrasts presented by the two sets of phenomena:

TABLE OF PULSE RATE AND TEMPERATURE.
(Thermometer in axilla.)

Case.	Pulse.	Temperature.
1	113 per minute.	102.0° Fah.
2	100 " "	100.0 "
3	112 full.	104.0 "
4	104 "	103.0 "
5	92 "	101.0 "
6	100 "	103.5 "
7	104 "	103.0 "
8	100 "	104.5 "
9	84 "	99.0 "
10	70 "	100.5 "

"From this table it will be seen that the highest pulse rate and the highest temperature did not correspond. On the contrary, the highest pulse at 113 had only a moderate degree of elevation of the thermometer, 102 degrees, while the pulse at 100 gave the highest temperature, and the lowest pulse rate, that at 70 beats per minute, was attended by coloration and an elevation of the thermometer to 100.5 degrees. There was, therefore, no constant uniformity of relation between the two sets of phenomena (pp. 270, 271).

Hemorrhagic Form:—I can testify to the statement, that in this form the yellow coloration constantly appears during the primary pyrexial state and while it is at its height, with the pulse over 100, and the thermometer in the axilla registering 101 degrees, 102 degrees, or even 104 degrees, the thermometer indicates generally a less considerable elevation of temperature in the hemorrhagic than in the sthenic cases. I have observed the pulse at 92 degrees and the thermometer at 101 degrees Fah. in a most characteristic well-marked hemorrhagic case. The pulse subsequently rose to 100, and 104 degrees in the same case, with elevations of the thermometer to 103.5 degrees and 103 degrees respectively, coloration of the skin being present to a marked extent. I do not, however, think that the temperature is generally so high in this class of cases. I believe that as a general rule the temperature in the hemorrhagic cases is from 2 degrees Fah. under that in the sthenic form. The transition from the sthenic to the hemorrhagic form is sometimes marked by a diminution in the rate and volume of the pulse, and a lowering of the thermometer; the patient's strength sinks rapidly at the same time, and in some cases a clammy sweat bedews the face for one or two days. This state of things is followed by, as it were, an explosive and universal lesion in the vascular system, leading to profuse hemorrhages at all available points of the cutaneous and mucous surfaces (pp. 274, 275).

"State of the Pulse:—I have nothing to record of a characteristic or peculiar kind in reference to the pulse. It is commonly less frequent than in the sthenic form, but has perhaps more of fullness and expansion, while its force is less. I have carefully noted the condition of the pulse in numerous cases, and in no one instance have I met with anything approaching to the dicrotous character either before or during the height of the hemorrhagic invasion; and that it was a very rare condition in the Lisbon epidemic is evidenced by the circumstance that in the large experience of Dr. M. Figueira, and of the physicians to the Desturo Hospital, it was presented but once; this observer could call to mind but one single case in which the pulse presented a well-marked dicrotous beat.

"The rate of the pulse is variable; it is commonly over rather than under, 100 per minute. It is most generally full, soft and responsive, and wanting in force and rebound. These, it will be observed are not very positive characters; but in such were presented in the great majority of cases observed by me.

"The pyrexial state is evidenced more decidedly by the state of the thermometer, which is usually over 100 degrees Fah. There is much variety in this respect; but what is important, and what can be positively stated, is that the hemorrhages occur during the pyrexial period, and with elevation of the pulse, and the thermometer above the standard. Thus we have seen them presented with characteristic force and intensity, while the pulse was at 100, and the thermometer in the axilla stood at 103.5 degrees; but this latter is unquestionably an exceptional elevation for the form of the disease now under consideration (pp. 376–377)."

My own investigations on the variations of temperature in different animals and in man under the action of various poisons and conditions of health and disease, and of climate, starvation and repletion, were begun in 1854, and have been continued without interruption up to the present time. The opportunities of recording observations on the pulse, respiration and temperature in various diseases were greatly enlarged during the recent Civil War (1861–1865). In 1870, a special monograph relating to yellow fever was transmitted to the AMERICAN MEDICAL ASSOCIATION, and in 1873 I endeavored to excite various observers in those cities in which yellow fever was prevailing, as in New Orleans, Memphis, Shreveport and Pensacola, to record critical observations on the changes of the pulse, respiration and temperature of the disease. The results of the portion of these labors which relate more especially to yellow fever will be found in the present chapter.

The temperature according to Schmidtlein,[38] is highest in the first few days of yellow fever, and very often reaches a height of from 40 to 41 degrees C., (104 to 105.8 degrees F.) Very frequently with slight evening exacerbations. From the fourth to the fifth day the temperature slowly falls, and sinks down to normal or even below this. In cases which end fatally, it rises again toward the end, some 2 degrees C. (3.6 degrees F.) or even more.

According to Dr. William N. Nixon,[39] during the recent fatal epidemic of yellow fever in Buenos Ayres, 1870–1871, a high range of temperature was rapidly attained, and maintained with the exception of differences of portions of a degree between the night and morning temperature, the night returns being slightly higher than those of the preceding morning; the decline was quite gradual.

I have refrained from citing the general observations of numerous writers, as Rush, Louis and a host of others, as to the presence of heat of surface in yellow fever, for they possess but little value for purposes of critical diagnosis, and confer little or no power of distinguishing between this disease and several other forms of malarial fever. We have sought rather to record the actual observations with the thermometer. A similar course will be pursued with reference to the changes of the pulse in this disease.

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[38] On the Temperature in Diseases, by Dr. C. A. Wunderlich, p. 405.
[39] London Medical Times and Gazetteer. 1871.

The fullest and most reliable observations upon the pulse in yellow fever, with which we are acquainted, are those given by Daniel Blair, in his account of the last yellow fever epidemic of British Guiana, 3d edition, London, 1852. According to this accurate observer: "The pulse was rarely very quick during any period of the disease. It was highest in the first stage, and gradually declined in frequency. Before death it generally became quicker and smaller, and when much fluid ejection occurred, it became extinct at wrist many hours before death. During convalescence the pulse was uniformly slow when no complication existed. The pulse was quickest in the cerebral variety. In some cases when the disease determined to the intestines, the pulse became startlingly slow, even on the second day of the disease; thus in Mr. Mackae's case it was 48; he recovered. In Case 2,895 of Seaman's Hospital, the pulse on the sixth day of fever was 24. The insidious nature of some of the attacks (when the seat of the malady was the intestinal, urinary or pulmonary apparatus) the perfect care of the patient, the external air of good health, and the *solemnity of the pulse* in such cases, frequently inspired the practitioner with a kind of awe and horror of the new, treacherous and remorseless malady.

TABLE SHOWING THE AVERAGE PULSE ON DIFFERENT DAYS OF THE DISEASE WITH THE NUMBER OF OBSERVATIONS FROM WHICH EACH AVERAGE IS OBTAINED.

No. of Observations.	Day of Disease.	Average Pulse.
121	1	97.40
338	2	90.80
406	3	83.53
388	4	80.44
311	5	78.56
206	6	78.74
125	7	78.78
71	8	75.62
46	9	75.76
29	10	77.58
16	11	76.37
7	12	76.00
5	13	79.20

In some cases of yellow fever, it seemed as if the poison acted directly, and at once, as a sedative on the heart; and in some cases there seemed a sudden and temporary excitability of it, which must have been favorable to the production of fatal local congestions.

Remarkable differences in the pulse are sometimes observed at different periods of the day.

1. Examples of rising in frequency:

Case 5,211. Morning, pulse 84; afternoon, pulse 126.

Case 828. Morning, pulse 80; afternoon, pulse 108.

Case 1,318. Morning of fifth day, pulse 70; afternoon, pulse 100.

Case 986. Morning of third day, pulse 72; afternoon, pulse 120.

Case 1,506. Morning of third day, pulse 52; afternoon pulse 86.

Case 2,053. Morning, pulse 100; afternoon of fourth day, pulse 150.

Case 2,215. Morning, pulse 112; afternoon of fourth day, pulse 156.

Case 2,215. Morning, pulse 92; afternoon of eighth day, pulse 134.

2. Examples of falling in frequency:

Case 583. Morning, pulse 108; afternoon, pulse 54.

Case 985. Morning, pulse 100; afternoon, pulse 72.

Case 986. Morning of second day, pulse 120; afternoon, pulse 108.

Case 1,137. Morning, pulse 96; afternoon, pulse 64.

Case 1,277. Morning of second day, pulse 100; afternoon, pulse 64.

Case 1,278. Morning of fifth day, pulse 120; afternoon, pulse 80.

3. Examples of the falling and rising suddenly:

Case 986. On second day falls from 120 to 108. On third day rises to 120 and again falls to 80.

Case 1,318. On fifth day rises from 70 to 100; falls to 80 on morning of sixth, and to 50 in evening.

Case 2,215. On fourth day rises from 112 to 156, and on eighth from 92 to 134.

The pulse is often observed to rise or fall suddenly in frequency shortly before death.

1. Examples of its rising:

Case 381. On day of death, rises from 68 to 80.

Case 656. On day of death, rises from 58 to 72.

Case 2,080. On day of death, rises from 88 to 120.

Case 2,601. From 80 day before death to 110 on day of death.

Case 2,609. On day of death rises from 78 to 100.

Case 2,680. On day of death rises from 84 to 120.

Case 2,712. From 80 day before death to 124.

Case 2,775. From 100, day before death to 134. From 80 to 96 and 116, on day of death.

2. Examples of its falling:

Case 367. From 130 to 98, day before death, and again rises to 120.

Case 656. From 84 to 54.

Comparison of adult pulses in convalescence from virulent yellow fever
with adult pulses in cases convalescent from inflammatory
diseases where no febrile action had existed. Made
in Seaman's Hospital Sept. 10, 1843.

YELLOW FEVER.

Name.	Pulse.	
Fletcher	50	Irregular
McLaren	56	Regular
Wilson	52	Regular
McTammany	58	Irregular
Allen	58	Regular
Total,	274	
Average pulse	54.4	

INFLAMMATORY CASES.

Name.	Pulse.	Previous Diseases.
Pease	115	Bronchitis, emphysema
Harding	102	Paracentitis
Hall	102	Asthma, empyema
Power	90	Paracentitis, cough
Tynas	100	Suspected tubercular infiltration in right lung
Total,	509	
Average pulse	101.8	

ACCIDENTS AND AILMENTS WITHOUT FEVER.

Name.	Pulse.	Previous Ailments.
Purcell	76	Muscular pain
Sinclair	84	Eccentricity of conduct
Martiu	84	Paralysis
Briau	84	Ankylosis of knee joint
Johnson	84	
Total,	412	
Average pulse	82.4	

NOTE.—It is to be observed that these remarkable differences in the frequency of the pulse are rarely accompanied by corresponding febrile exacerbations. Although the remarkable slowness of the pulse in convalescence from yellow fever was early observed, the following limited trial for comparison by me on the subject is believed the only one which has been made. The fact was observed before the experiment was instituted.

Dr. Davy remarks upon these observations in his notes to the work of Dr. Blair: "Slowness of pulse in connection with certain diseases of the abdominal viscera—not of an inflammatory kind, is worthy of remark. Jaundice is a striking instance of this kind; less marked ones are met with in cases of functional derangement of the primæ viæ, though not without exceptions." . . "Unusual slowness of pulse was often observed in the yellow fever of Barbadoes, and commonly accompanied with undue coolness of skin, especially in the extremities; it was a remarkable feature of the disease after its first invasion,—the time varying in different cases. The author's private observations on this symptom appear to me to be very valuable (pp. 74-77). Recent observations in New Orleans have confirmed the accuracy of Dr. Blair's statement with reference to the slowness of the pulse in yellow fever—as will be seen by an examination of the following table—which includes the labor of several observers working wholly independent of each other.

Dr. Porcher of Charleston, Dr. Charles Faget,[40] Dr. Just Finaste, Thomas Layton,[41] Prof. Samuel Logan,[42] Drs. Saunder[43] and Mitchell[44] of Memphis, have recorded valuable observations on the changes of the temperature, pulse and respiration in yellow fever.

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[40] New Orleans Medical and Surgical Journal, 1873.

[41] Reply to the Inquiries of Prof. Jos. Jones, of New Orleans, relating to the Temperature of Yellow Fever.—New Orleans Medical and Surgical Journal, March, 1874, p. 695.

[42] Bedside Notes on the Pulse, Temperature and Urine in Cases of Yellow Fever, Observed at Pensacola Navy-Yard, in the fall of 1874. —New Orleans Medical and Surgical Journal, May, 1875. p. 779.

[43] Observations on the Yellow Fever Epidemic of 1873, at Memphis, Tenn. By D. D. Saunders, M.D. Prepared in response to the request and inquiries of Prof. Joseph Jones, M.D., of New Orleans.—New Orleans Medical and Surgical Journal, May, 1874, p. 791.

[44] Yellow Fever in Memphis in 1873. By R. W. Mitchell, M.D., of Memphis, Tenn.—Rich. and Louisville Med. Jour., May, 1874.

Dr. Charles Faget,[45] of New Orleans, who is well known to the profession in this country and in Europe, for the accuracy of his observations upon disease and for the depth and extent of his learning, has produced one of the most striking and valuable articles on the relations of the temperature and pulse in yellow fever.

Dr. Faget bases his conclusions on thirty-eight tables of cases, recorded during the yellow fever epidemic of 1870 in New Orleans, furnished as follows: Sixteen by Dr. Touatre, Physician to the French Asylum; some from the asylum, the rest from the city; seven by Professor Bemiss, all from the Charity Hospital, thermometer in the axilla; three by Dr. Layton, and twelve by himself; the last fifteen from the very focus of the disease. Drs. Layton, Touatre and Faget took the temperature in the patient's mouth, placing the thermometer between the gums and cheek. The main object of this article as announced by its author "is to prove that yellow fever is a fever of a *continuous* type, and that it takes its place as a *specific* fever, distinct from all other forms, and is especially to be distinguished from the *malarial species.*"

Dr. Faget thus places himself in accord with a host of writers on yellow fever, from the time of Lining, of Charlestown, S. C., in 1754, to the present moment, including the names of Bally, Louis, Blair, Bennet Dowler and John Henderson, of New Orleans; and in opposition to the school founded by Bancroft and ably supported by Chisholm and his latter day followers who confounded true yellow fever with certain forms of malarial fever.

None of the tables in the article of Dr. Faget support the assertion in the article, "Fevers," of the new

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[45] Type and Specific Character of True Yellow Fever, as shown by Observations taken with the Assistance of the Thermometer, Science and Watch. By J. C. Faget, Member of the Société Médicale d' Observation. New Orleans Medical and Surgical Journal, 1873. The Type and Specificity of Yellow Fever, established with the aid of the Watch and Thermometer. By Dr. J. C. Faget of the Faculty of Paris. New Orleans and Paris, 1875.

French Dictionary of Medicine (1871): "In some fevers the remission is long and complete, as in yellow fever, for instance, in which the *initial attack* is separated from the *terminal fever* by a *remission* of one or several days duration." (Vol. XIV., p. 742.) On the contrary, according to Dr. Faget, "yellow fever shows *one single effervescence*—only *one paroxysm*, never a true remission, and the *unique attack* subsides immediately. Its progress is regressive as soon as it appears. This last peculiar feature would not show as plainly in our tables, but for the fact that they often begin after the second and even third day. The fact is that the decline is marked from the very beginning, in the line of the fever only; as for the temperature we shall see that it even increases, at least, in two-thirds of the cases during the first two or three days. . . . The thirty-eight observations of 1870 also contradict the opinion concerning the *short duration* of yellow fever. According to La Roche's opinion, based on immense learning, it would be three days (3×24=72): A febrile stage of about seventy hours' duration, more or less, is succeeded by a period of complete cessation of fever (Vol. I., p. 426). Judging from our tables, it should be six or seven days (6×24=144 hours): "The maximum of temperature in yellow fever, in New Orleans in 1870, has been, in the average of cases, 104.1 degrees. Such is the average maximum of fevers in general; there is, therefore, in this, nothing peculiar in yellow fever. The particular and special feature is that: 1, this average maximum of temperature is very rapidly reached in yellow fever cases. In about one-third of our cases (twelve in thirty-eight) the maximum has been reached on the very first day; in one-fourth of the cases (nine in thirty-eight) it was on the second day; in another third of cases (fourteen in thirty-eight) on the third day; and in three cases the disease had gone over three days before reaching its maximum, and it went on steadily decreasing till it reached the normal standard, and even below that point, except in cases where special circumstances or accidental causes easily accounted for, such as secondary congestions appeared. Thus we can say that there exists no stationary stage in yellow fever. The decrease of the temperature has been remarkably slow. Up to the fifth day it had not yet reached the normal standard in most of our cases. The exceptions were such as were very mild, or such as proved rapidly fatal. In twenty or thirty the thermometer registered 100 or 102 degrees on the fifth day. . . .

"In short, the march of the temperature in our tables is marked: 1, by a period of increase, very rapid and of very short duration; and 2, by a period of decrease, rather slow and long. There is no sign of a true stationary stage. Thus the study of temperature in yellow fever offers for our consideration only an effervescence and a defervescence. . . .

"I shall sum up in saying that, the march of the temperature in yellow fever, as shown by the graphic lines representing it, is characterized by a unique paroxysm with an effervescence of one to three days, followed by a defervescence of four to seven days, without any stationary stage. . . .

"We consider that we are now authorized to say that in yellow fever we are able, in the majority of serious cases, to detect from the beginning of the fever, one of its most precious diagnostic signs, and that is the divergence of the lines indicating the march of temperature and pulse. The line of the pulse descends immediately, while the line of temperature ascends, and that during one, two or even three days. . . .

"But I shall insist upon saying that the direction of the lines of temperature and pulse is of special importance in the incipient stage of the febrile paroxysm of yellow fever, in view of the diagnosis of the disease. In the great majority of serious cases, the line of pulse descends, while the line of temperature ascends. Are we not justified in our belief that there very likely exists no other disease in which the same occurrence obtains? . . .

"Let us now consider the specific character of yellow fever, which shows itself in our opinion, by the action of its morbific principle on the heart, this action being denoted by a diminution in the number of pulsations from the very beginning.

"In the work that I published on yellow fever in 1859, after having studied the progress of the fever, without the thermometer, guided only by statistics of the pulse, and those statistics compiled from at least a hundred observations, taken by a dozen physicians during the epidemics of 1839 , 1853, 1858, I expressed myself in the following manner (p. 85):

"In all these observations written at the bedside of the patients during three different epidemics in New Orleans we perceive, nearly without exception, that the pulse at its *apogeum*, from the first day gives even more than 100, sometimes 110 and 120 pulsations; begins to fall the second day, continues to decrease regularly the third, and gives from that time 70 to 80, sometimes much less."

"The regular and rapid decrease of the pulse is such in yellow fever, from a record of a hundred observations, that we could recognize it as the true characteristic of that fever.

"To-day, after the epidemic of 1870, that is to say, after having been able to study the progress of yellow fever, not only with the 'independent second watch,' but at the same time with the thermometer, I think further hesitation is impossible; the diminished frequency of the pulsations, that is to say, the heart's action, show themselves from the beginning in yellow fever, and consequently in the height of febrile excitement, as certified by the thermometer. This is the essential characteristic of this fever, and, in fact, a like decline of the pulse is not a simple abatement of the fever, since it occurs in the great majority of cases during the increase of temperature. We find, then, a diminution of the beats of the heart, produced by a direct specific action of the poison itself of yellow fever, in the central organ of circulation. This specific action of yellow fever poison on the heart can be compared to the effect produced on that organ by certain poisons, as, for instance, digitalis and veratrum viride. . . .

"Now the specific character,clinically demonstrated has just received the most remarkable anatomic confirmation in the observations upon yellow fever (just published by Prof. Joseph Jones, of the University of Louisiana). This is what he says on page 8 of his pamphlet: "The central organ of circulation is structurally altered and enfeebled in yellow fever, the muscular structures of the heart present alterations similar to those observed in the liver and kidneys. Oil, granular, albuminoid, or fibroid matter is deposited within and around the muscular fibrillæ, and the organ after death presents a yellow flabby appearance. These lesions of the heart, shown by careful *post-mortem* examination, are characteristic of the disease." . . . . The general table of the average of the pulse, furnished by Blair, like ours of New Orleans, not only shows the steadily decreasing character of the pulse in yellow fever, but it also demonstrates beyond a doubt that this fever has but one paroxysm, and consequently it is a continued fever, continually decreasing but still continued. . . . . "If, then, yellow fever remained a fever with but one paroxysm, in Guiana as well as in New Orleans, it proves it not only to be a continued fever, but one that resists efficaciously the concomitant influences of malarial fever.' (Type and Specific Character of True Yellow Fever, *New Orleans Med and Surg. Journal*, September 1873, pp. 145–168.)

Dr. D. D. Saunders, of Memphis, Tenn., in his valuable "Observations on the Yellow Fever Epidemic of 1873 at Memphis, Tenn.," gives a tabulated record of the pulse and temperature in seventy-three cases of yellow fever. The daily average of the temperature and pulse, was as follows: First day temperature 102 degrees; pulse 114½. Second day temperature 102.5 degrees; pulse 112½. Third day temperature 102.2 degrees; pulse 107¼. Fourth day temperature 102 degrees; pulse 96½. Fifth day temperature 102.2 degrees; pulse 86½. Sixth day temperature 101.5 degrees; pulse 74. Seventh day temperature 99.5 degrees; pulse 69¼. Eighth day temperature 99 degrees; pulse 68. Ninth day temperature 97.8 degrees; pulse 64⅔. Tenth day temperature 98.1 degrees; pulse 65½. Eleventh day temperature 98 degrees; pulse 67¼. Twelfth day temperature 98.2 degrees; pulse 71⅔. Thirteenth day temperature 98.5 degrees; pulse 80. Fourteenth day temperature 98.5 degrees; pulse 82.

In ten fatal cases the daily average of the temperature and pulse was: First day temperature 105 degrees; pulse 120. Second day 105.5 degrees; pulse 120. Third day temperature 105.7 degrees; pulse 116½. Fourth day temperature 106.5 degrees; pulse 114½. Fifth day temperature 104.6 degrees; pulse 106⅔. Sixth day temperature 103 degrees; pulse 100⅗. Seventh day temperature 102.8 degrees; pulse 99. Eighth day temperature 101 degrees; pulse 99⅛. Ninth day temperature 97.5 degrees; pulse 81. Tenth day temperature 99 degrees; pulse 100. Eleventh day temperature 98 degrees; pulse 120. Twelfth day temperature 97 degrees; pulse 130. Thirteenth day temperature 96 degrees; pulse 130.

Of the ten fatal cases, five died on the fifth day of uremia; one died on the sixth day, of uremia; one on the seventh day, of nervous prostration; one on the ninth day, of uremia; one on the tenth day,of uremia; and one on the fourteenth day, of uremia. No case of uremia lived over fifty hours, after urinary suppression set in.

Dr. Saunders remarks: "The tables accompanying this report show yellow fever to be a full fledged fever within a few hours after its initiation, and the circulation is as rapid then as it ever gets to be unless complications arise, as in the winding up of the fatal cases. The pulse begins to diminish in frequency within a few hours of the beginning of the fever, and in the stage of convalescence will descend below the normal standard. I saw it descend to thirty-six beats per minute and the patients recovered. The temperature, however, will generally either hold its starting point, or gradually ascend from 72 to 120 hours, and then if there are no complications, begin a gradual descent with the pulse, and go below the normal standard of 98.5 degrees in convalescence. I saw the thermometer descend below 95 degrees, and the patient recover. In these cases when the pulse and thermometer descend gradually together, after the second day, no apprehension of danger need be entertained; they will always get well. Such was the result of my observation. . . . . I saw no case of yellow fever recover when the thermometer marked over 106 degrees, on the first and second visits, and I saw no fatal case when the pulse and the thermometer gradually descended together below the normal standard.

"I believe with Prof. Joseph Jones, that when the temperature ranges very high from the first, the patient may die suddenly, overpowered by the poison, as in cases of sunstroke."

The results obtained by Dr. R. W. Mitchell,[47] of Memphis, Tenn., and by Prof. Samuel Logan[49] at Pensacola, sustained those previously announced by Blair and others. Dr. Mitchell gives a valuable table of the daily variations of the pulse and temperature in forty-eight cases of yellow fever, the general results of which will be more fully examined hereafter.

With this general historical introduction we will proceed to present specific details and the results of original investigations. The general results of my investigations upon the changes of temperature and conditions of the pulse in yellow fever may be formulated thus:

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[47] Yellow Fever in Memphis in 1873, by R. W. Mitchell, of Memphis Tenn., Richmond and Louisville Medical Journal May, 1874.

[49] Bedside Notes on the Pulse, Temperature and Urine, in cases of Yellow Fever observed at Pensacola Navy Yard, in the fall of 1874, New Orleans Medical and Surgical Journal, May 1875, p. 779.

Note—New Orleans Medical and Surgical Journal, 1874, p. 791.

The maximum elevation of temperature is attained upon the first, second, and third days of the disease; ranging, according to the severity of the attack, from 102 degrees to 110 degrees in the axilla; and, as a general rule, from the third to the fifth day steadily falls and sinks down to the normal standard, and even below. In some fatal cases it rises again toward the end, rarely, however, reaching or exceeding, during the stage of passive hemorrhages, black vomit, jaundice, and urinary suppression, 104 degrees, and, as a general rule, never attains the high degree of temperature characteristic of the first stage. The supervention of an inflammatory disease, or the occurrence of an abscess, or the access of malarial fever, after the first stage, may in like manner cause a progressive elevation of temperature, with slight evening exacerbations.

The pulse at the commencement of the attack is rapid and full. The frequency of the pulse does not, however, as a general rule, continue to correspond with the elevation and oscillations of temperature, as in many other febrile diseases; and in many cases of yellow fever the remarkable phenomenon is witnessed of the pulse progressively decreasing in frequency, and even descending below the normal standard, while the temperature is maintained at an elevated degree; and on the other hand the pulse frequently increases in frequency, but diminishes in force, near the fatal issue. The occurrence of copious hemorrhage from the stomach or bowels may be attended with sudden depression of temperature, and increase in frequency with diminution in the force and fullness of the pulse.

The remarkable progressive decrease in the beats of the pulse after the first stage in many cases appears to be due to several causes; as the anatomic changes in the heart (acute fatty degeneration), and the retention in the blood of the bile and urinary constituents.

If the temperature rises in the first stage above 105 degrees, the patient is in imminent danger; and if it reaches from 107 degrees to 110 degrees, death is inevitable, whatever may be the treatment adopted. In cases attended with the rapid rise of the temperature to 106 degrees and beyond in the first stage, death sometimes occurs suddenly, and apparently solely from the effects upon the blood and nervous system of the great elevation of temperature, as in sunstroke. The truth of this statement will best be illustrated by actual observations, and especially by the following case, which has recently occurred in my private practice:

Mrs. W., aged 28 years, has resided in New Orleans eighteen months; large, well-developed, with clear complexion and high color in health. Mrs. W. called at my office July 21, stating that she had just passed through the menstrual period, which had been protracted for ten days, was very profuse, and had confined her to bed. She complained of great weakness, "heaviness" of feeling, vertigo, and pain in the head, back, and limbs, symptoms which appeared to be attributable, at least in part, to the hemorrhage, as I had attended her upon previous occasions when suffering with analogous symptoms resulting from profuse menstruation. July 22, 1 P.M., I was called to Mrs. W., and found her suffering with slight febrile excitement; pulse 90, full and strong; face flushed; pain in head, back and limbs. July 23, 9 A.M. Face greatly flushed and of scarlet hue; capillaries of the extremities and face and surface generally congested; patient greatly agitated and alarmed: says she has yellow fever, and will surely die. It was difficult, if not impossible, to calm her fears. Skin warm, but bathed in profuse perspiration; pulse 108, full and strong; great pain in back and head. 3:30 P.M., pulse 110; temperature 103.5 degrees; urine abundant, light yellow, slightly turbid from presence of vesical and vaginal mucus; a trace of albumen. Menstrual flow returned for an hour or two during the morning, but ceased again; capillaries of the surface intensely congested; tongue red at tip and edges, furred in center. July 24, 9 A.M., pulse 118, respiration 30, temperature 106.8 degrees; skin hot and dry. The fever rose in the evening; patient talked and muttered in her sleep, and frequently awoke suddenly with a start and cry; moans and sighs with every breath; is greatly agitated and alarmed; declares the case is utterly hopeless, and that her lower extremities feel as if they were paralyzed. Pain in head, back, and limbs intense; nausea constant and distressing, but no vomiting; heavy, disagreeable odor emitted by the body, as in yellow fever; great congestion of capillaries; face and hands of a scarlet hue; an eruption has appeared upon the forehead; the surface of the face, trunk, and extremities is as highly injected and as red as in scarlet fever or measles, but the brilliant redness is more uniformly diffused over the surface than in either of these diseases. Urine abundant; light-yellow color; specific gravity 1020; contains a small amount of albumen, with detached cells from the tubuli uriniferi and yellow granular casts, together with vesical and vaginal epithelium. Upon standing, the urine let fall a moderate deposit of urates of ammonia and soda, mingled with the cells and casts. 3 P.M., pulse 108, respiration 36, temperature 106.3 degrees; skin hot and dry; face and surface generally of a deep scarlet hue; pressure drives out the blood from the capillaries and leaves a white spot, into which the blood slowly returns. Tongue coated in center, with yellow fur and red at tip and edges; swollen, with margins indented by the teeth; moist and soft. Nausea, depression, fear of death, and bad odor from the body unchanged. 9 o'clock P.M., pulse 112, temperature 106.2 degrees, respiration 38; moans and sighs with every breath; when spoken to answers rationally, but often slumbers, starts, and jumps in a delirious, nervous manner; odor from the body heavy and offensive.

July 25 10 A.M.. condition unchanged; pulse 118; full and strong; respiration 36; temperature 108 degrees. The delirium and restlessness of the patient prevented thermometer being held well in the axilla; the actual temperature was therefore somewhat above 108 degrees, and probably reached from 110 degrees to 113 degrees in the cavities of the heart. Conjunctivæ congested; gums red and spongy; odor of body very offensive; surface of face, trunk, and extremities greatly congested and of a brilliant scarlet hue; forehead covered with a distinct papular eruption. Urine light colored; specific gravity 1020; contains albumen in considerable amount, numerous granular casts of tubuli uriniferi, kidney cells, granular yellow albuminoid matters, and amorphous deposits of urates of ammonia and soda. Two hours after I was summoned to the bedside of the patient, and found her in *articulo mortis;* pupils contracted; spasmodic respiration, with death rattle in throat. I was informed that she had started suddenly in a disturbed sleep, made several ineffectual efforts to vomit, and passed immediately into this state. She was unable to swallow. Sinapisms were freely applied, but without effect.

The treatment of this case will be briefly mentioned, although we are not now engaged with this subject. The bowels were first opened with a saline cathartic, followed by quinin. Rest was then promoted by Dover's powder. The bowels were afterward kept open by means of enema. The diet was light but nutritious, and administered in small quantities at regular intervals; the action of the kidneys was promoted by the use of gentle diuretics, as orange-leaf tea and water charged with carbonic-acid gas. Quinin aggravated the delirium, and was abandoned at once. Alcoholic stimulants were found in like manner to aggravate the pain in the head, the nervous excitement, and delirium. Cold wet cloths were applied to the head, and the surface bathed with tincture of camphor and water.

In the last moments of life the scarlet flush of the surface gradually faded, and at the moment of death, which occurred at 1 P.M., the surface presented a yellow jaundiced hue; after death, body mottled; decomposition rapid. I attributed the death of this patient to the high degree of heat and the consequent disorganization of the blood and derangement of the nervous and muscular forces consequent upon the action of the febrile poison.

Case 2.—Yellow fever: high temperature, 106.5 degrees in axilla; suppression of urine; black vomit; recovery; treatment, ice water injections to rectum; application of ice to epigastrium; sulpho-carbolate of sodium internally. Dominick Polmisano; age 18; native of Italy. Has resided in New Orleans six years; residence St Anne Street near levee. Black hair, black eyes, dark complexion. Had a slight chill, followed by pain in the head and back and fever. Oct. 14, 1873. Entered Charity Hospital, October 16, at 4:30 P.M. At this time the patient had high fever; hot skin; face very red with great capillary congestion of the surface generally. Capillaries of conjunctivæ, greatly congested and of a yellowish tinge. Body emits an offensive smell. Breath very offensive. Tongue red at tip and edge and coated with light yellow fur shading into brown in the center and root of the tongue. Gums very red. Patient complains greatly of severe pains in heart, epigastrium, lumbar region and lower extremities. At 8 P.M., pulse 106; respiration 28; temperature of axilla 104 degrees.

℞. Potassii bromid . . . . ʒij
Liquid ammoniæ acetates . . . . f℥iv.
Mix: Tablespoonful every four hours.

October 17, 8 A.M., pulse 100; respiration 28; temperature of axilla 106.5 degrees. Patient restless; complains of intense pain in the head, back, epigastrium and extremities. Nausea. Great congestion of capillaries of face and extremities. Skin presents the appearance of a purplish and scarlet rash. Conjunctiva injected and jaundiced. Tenderness upon pressure over epigastrium. Tip and edges of tongue red, coated in center; gums red. Has passed no urine since entering the hospital; when the catheter was introduced, only one fluid ounce of urine was drawn off, which upon chemic and microscopic examination was found to be loaded with albumen and bile, and casts of the tubuli uriniferi and excretory cells of the kidneys. To control the nausea, an ice bag was placed over the epigastrium. For the suppression of the urine, ice cold water was thrown up the rectum in large quantities. The injections of ice water had the desired effect, and caused the excretion and evacuation of a considerable amount of urine, which was heavily loaded with albumen and granular casts. In order to prevent septic changes in the black vomit if it should be formed, I ordered the following:

℞. Sodii, sulpho carbolate . . . . ʒi

Divide into three parts, one part (20 grains) dissolved in water, every three hours. Brandy and carbonic acid water. Ice and milk, internally in small quantities at regular intervals. Small fragments of ice to be held in the mouth.

At 8 P.M., condition unchanged; jaundice and nausea and restlessness increasing; urinary excretion very scarce. Urine contains albumen, bile and granular casts and cells of tubuli uriniferi. Pulse 98; respiration 26; temperature of axilla 104.1 degrees. Continued ice bag to epigastrium; ice water enema; sodium sulpho carbolate; iced milk and brandy diluted with carbonic acid water.

October 18, 4 A.M., the patient has begun to throw up black vomit. Continued to eject black vomit at short intervals quite freely until 8:30 A.M. The sulpho-carbolate of sodium appears to have exerted some beneficial effect, as the black vomit is of a brighter color than usual and contains coagula of blood.[50] At 8 A.M., pulse 82; respiration 28; temperature of axilla 102.1 degrees. The supervention of black vomit has been attended by a reduction of the temperature. Great congestion of peripheral capillaries. Jaundice; gums red;

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[50] These results appeared also to be due to the local application of ice to the epigastrium, and the injection of ice-cold water into the rectum, which not only controlled the nausea, but also reduced the excessive temperature.

tip and edges of tongue very red, center and body of tongue coated with yellow and brownish-yellow fur. Blood oozed from tongue and gums. A small quantity, about half a fluid ounce, of urine drawn off by catheter. Urine loaded with albumen, casts and excretory cells; also contains bile. Patient delirious and very restless. A strong man was ordered to sit by his bed, to keep him as quiet as possible and carefully covered, and to prevent him from rising and getting out of bed.

℞. Sodii sulpho-carbolate . . . . ʒij.

Divide into six powders; one powder every three hours.

Inject one pint of ice-cold water into the rectum, at intervals of four hours, until the kidneys act freely. Apply ice-bag to epigastrium. Apply sinapisms to back of neck and lumbar region over seat of kidneys. Administer at regular intervals of two hours, alternately, small quantities of brandy largely diluted with water charged with carbonic acid, and iced milk. Fragments of ice to be placed in the mouth. The brandy and carbonic acid water appeared to increase the nausea and vomiting, and were accordingly discontinued; the sodium sulpho-carbolate, on the other hand, appeared to quiet the stomach, and during the day the black vomit ceased and the patient enjoyed several hours of refreshing sleep. At 6 P.M., pulse 80; respiration 24; temperature of axilla 102 degrees. Patient in much the same condition but not so restless. The injections into the rectum of the ice-cold water were followed by a pretty free evacuation of high-colored albuminous urine. Black vomit has ceased but nausea still continues. Hemorrhage from the tongue and gums still continues. Continue treatment.

October 19. Condition of patient still alarming; great congestion of capillaries; the face presents a deep mottled purplish appearance; hemorrhage from gums and tongue. No return of black vomit. Kidneys excreting urine more freely. Pulse 94; respiration 24; temperature in axilla 100 degrees. Continue sulpho-carbolate of sodium, 20 grains every three hours; also ice-water injected occasionally if kidneys do not act freely. Iced milk at regular intervals; also small quantities of borax and water charged with carbonic acid. At 7 A.M., patient apparently improving; intellect clearer; black vomit has ceased; nausea relieved; capillary congestion less; jaundice not so marked. The ice-water enema caused a free discharge of high-colored albuminous urine. Pulse 76; respiration 20; temperature of axilla 100 5 degrees. Continue sulpho-carbolate of sodium, ice-water enema, iced milk and brandy and carbonic acid water; reapply ice-bag to epigastrium; nourishment of the patient has up to the present time been confined chiefly to iced milk.

October 20, 8 A.M., patient decidedly better; vomiting has ceased; no nausea; free secretion of urine; capillary congestion disappearing and jaundice less intense; pulse 68; respiration 18; temperature 99.5. From this date the improvement of this patient was steady, and on October 22 the capillary congestion had almost entirely disappeared and the coloration of the skin was far less intense. Patient complains that he does not get enough to eat; says that he feels "bully," and putting his hand on his epigastrium says that his "*belly is hungry, and cries all the time for something to eat.*" Continued to improve and was discharged. The following is the record illustrating the relations of the pulse, respiration and temperature in this case:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1873	M.	E.	M.	E.	M.	E.	
Oct. 16		106	. .	28	. . .	104 °	Fever.
" 17	100	98	28	26	106.5°	104.1°	Jaundice, capillary congestion, albumen and casts in urine.
" 18	82	80	28	24	102.1°	102 °	Urinary suppression, black vomit, hemorrhage from gums.
" 19	94	76	24	20	100 °	100.5°	Black vomit, less urine, scant hemorrhage from gums.
" 20	68	70	18	24	99.5°	100.1°	Vomiting arrested, urine more abundant.
" 21	66	64	20	22	100 °	100 °	Improving, hungry, urine abundant.
" 22	62	60	20	19	99 °	99.8°	Continues to improve.
" 23	62	66	20	24	98.9°	99.4°	Convalescent, sitting up, cheerful, bright expression of eyes.

The restoration to health was rapid and complete.

Case 3.—Yellow fever; sudden rise of temperature; treatment, sulpho-carbolate of sodium and quinin; recovery. Wm. H. Williams; native of New Orleans; age 26. Entered Charity Hospital, Ward 27, on the night of Nov. 8, 1873. Patient states that he has been at work for some two months near the city on dredge boat, cleaning out a canal. On the previous day had a chill, followed by high fever and delirium, and in this condition was brought to the hospital. The patient was ordered tablespoonful of the liq. ammoniæ acetates, every three hours. November 9, patient is more rational and complains of intense pain in the supraorbital region, along the spinal column and in the thighs and knees. Conjunctiva of eyes congested. Considerable capillary congestion of surface with marked jaundice. Gums red, inflamed and bleeding; tongue red at tip and edges, furred and fissured in center and bleeding. Body emits a disagreeable odor. M., pulse 84; respiration 30; temperature 98.5 degrees. Ordered 20 grains of the sulpho-carbolate of sodium, every three hours. Evening, patient in much the same condition but restless. Temperature 100.5 degrees; pulse 96; respiration 27.

November 10, urine scanty and loaded with albumen and casts of tubuli uriniferi. Pulse 80; respiration 30; temperature 101 degrees. Ordered continuance of sulpho-carbolate of sodium; also ice-water injections into the rectum. Evening, pulse 144; respiration 40; temperature of axilla 106 degrees. Urine scanty and loaded with albumen; high fever; vomiting. I was unable to account for this sudden rise in the temperature.

November 11, fever has subsided and the patient appears to be much better. M., pulse 96; respiration 36; temperature 98 degrees. Evening, pulse 93; respiration 30; temperature 98.5 degrees. Ordered 10 grains of quinin every three hours. Continue sulpho-carbolate of sodium. 20 grains every four hours. Milk punch and whisky diluted with carbonic acid water.

November 12, pulse 96; respiration 36; temperature 98 degrees. Evening, pulse 108; respiration 34; temperature 99 degrees.

November 13, M., temperature 101 degrees; pulse 78; respiration 24. Evening, temperature 99.5; pulse 78; respiration 24.

November 14, patient improving. M, temperature 97.5 degrees; pulse 69; respiration 21. Evening, temperature 97 degrees; pulse 63; respiration 20. Continue quinin 5 grains three times daily, also sulpho-carbolate of sodium.

November 15, M., pulse 60; respiration 18; temperature 96.5 degrees. Evening, pulse 60; respiration 20; temperature 97 degrees.

November 16, patient improving rapidly; albumen has disappeared from the urine. Temperature 98 degrees; pulse 60; respiration 22. Continued to improve. Discharged December 12.

Case 4.—Yellow fever; high temperature, 106.5; jaundice; slow pulse; treated with sulpho-carbolate of sodium, 20 grains every four to six hours; sulphate of quinia; ice-water injections and brandy diluted with carbonic acid water and iced milk. Recovered. Tully Brown; stout strong man; large muscles; brown hair; dark eyes. Has been in America five years. Has been working on the Mississippi River during the past year; occupation laborer. Patient states that he was seized with a chill Oct. 25, 1873, at 9 P.M. The chill and fever returned October 26 and 27. Admitted into Charity Hospital, Ward 24, Oct. 29, 1873. The following is the record of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1873	M.	E.	M.	E.	M.	E.	
Oct. 29	88	96	20	28	106.2°	106.2°	Some pain in head and back; skin hot and pungent; peripheral blood vessels congested; conjunctiva congested; bowels constipated; tongue red at tip and edges, and coated with brown fur in center; ℞. Quinia sulph. ℈i; divide into two parts, one every three hours.
" 30	52	90	20	28	100.5°	101.2°	The quinin, 20 grains, appears to have reduced temp. and frequency of pulse, but not permanently; patient sweating profusely and general appearance imp'd; tongue less red and coated. Continued quinin; chill at 10.30 A.M.
" 31	78	92	25	26	106.5°	105°	Urine scant and loaded with albumen and casts, restless, complains of pain in head and back; jaundice; nausea; slept but little during night; quinin; sulph. carb. of sodium and ice-cold enema to bowels; ℞. Quinia sulph. ℈iss; pulv. rhei, ℈i; divide into three parts; one every three hours.
Nov. 1	74	78	24	28	101.5°	102.5°	Patient no better; not so restless; urine less abundant; ice-water enema appears to have been beneficial; less nausea; skin yellow; capillary congestion of extremities; albumen and casts in urine; continued sulpho-carb of sodium; urine suppressed; complains of pain in abdomen; repeated ice-water enema.
" 2	76	76	24	24	101.8°	101.8°	Restless; pain in head and abdomen; urine albuminous but more abundant; continued sulph..carb, of sodium.
" 3	84	72	24	22	102°	102°	Dull countenance; slept but little during past three nights; bowels constipated; urine scant; jaundice; capillaries congested; quinia and sulpho-carb. of sodium.
" 4	59	70	18	20	100°	98.5°	Improving; urine more abundant; ice-cold enema appears to have been beneficial.
" 5	64	56	22	20	99.8°	98.8°	Slept well during night; in profuse perspiration; albumen diminishing in urine, which is more abundant.
" 6	62	58	18	20	100°	98.8°	Albumen disappeared from urine; continues to improve; passed about 32 fluid ounces urine in last 12 hours.
" 7	. .	62	. .	18	. . .	100.5°	Countenance more natural; improving.
" 8	60	. .	20	. .	99.8°	. . .	Tongue moist and clearing off; appetite good; rests well; skin moist; steady improvement.
" 9	60	54	25	18	100.2°	99.5°	Improving.
" 10	62	66	20	18	98.8°	100°	Convalescent.
" 11	60	62	18	20	99.5°	99.8°	
" 12	64	60	16	16	99.8°	99.5°	
" 13	62	76	20	20	100°	98.8°	
" 14	60	. .	17	. .	99.8°	. . .	Discharged November 15.

Case 5.—Yellow fever; jaundice and slow pulse; urinary suppression relieved by ice-water injections; sulph-carbolate of sodium internally. Adolph Kammer; age 21, native of Sweden, has been in America six years, in St. Louis, from which place he came to New Orleans. Resided at 153 Levee Street, near the French Market. Had been in New Orleans one week, when he was attacked with fever. Entered Charity Hospital, Oct. 1, 1873, Ward 13, on second day of disease. Tongue red at tip and edges, and furred in the center; gums red; conjunctiva congested; skin hot and dry; patient restless and complains of intense pain in the head and back. The following is an outline of this case:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1873	M.	E.	M.	E.	M.	E.	
Nov. 2	84	82	32	43	104.5°	105°	Skin hot and dry; slight jaundice; gums congested; tongue dry and swollen; kidneys acting imperfectly. Although temp. in evening reached 105°, pulse was only 82.
" 3	86	96	32	34	102°	102.5°	Passed a small quantity of urine by the catheter; urine loaded with albumen and contained many yellow granular casts; resp. rapid and irregular, and patient appears in great danger; ice-water injections per rectum appeared to do good; caused excretion of some urine; in evening complained of pain in epigastrium, which was relieved by ice bag to epigast'm.
" 4	72	70	28	36	101.5°	102°	During night hemorr. from gums; passed urine quite freely towards morning; urine dark colored and loaded with albumen and bile; on 3d patient was placed on sulpho-carb. of sodium, 20 grs. every 4 or 6 hours; milk punch, brandy and beef tea and fragments of ice in the mouth.
" 5	54	48	26	24	98.5°	101°	During 4th perspired freely and appeared to be improving; became much more quiet; sedative water to the surface appeared to quiet him greatly; on 5th passes urine freely, looks more cheerful and is improving; jaundice well marked but less.
"	52	50	26	22	98.3°	98°	Passed a very comfortable night; jaundice disappearing; urine abundant; still contains albumen, but amount is diminishing.
" 7	46	50	22	24	97.5°	98.5°	Sitting up; all pain has disappeared; complains of nothing but a cough, which at times annoys him; appetite good; sleep quiet and refreshing; jaundice disappearing.

Nov. 8	45	54	22	23	97.7°	98°	During coughing spat up some dark blood; as there was no congestion of lungs and no rise of temperature, the blood was supposed to have come from mouth and throat. Albumen disappeared from urine,
" 9	52	50	26	27	97.8°	98°	Is up and walking in ward. Continues to improve.
" 10	48	48	20	19	98°	98.2°	Continues to improve.
" 11	52	52	26	24	98°	98.7°	Jaundice disappearing rapidly, very slight tinge in skin and traces of yellow in eyes.
" 12	54	52	24	24	98°	98.2°	Gains strength rapidly.
" 13	52	46	26	24	98°	98.2°	Discharged from hospital Oct. 17.

Case 6.—Yellow fever; jaundice; urinary suppression; delirium; treatment, ice-cold injections; sulph-carbolate of sodium; urinary excretion reëstablished; recovery. John Pflum; age 26; native of St. Louis; laborer. Entered Charity Hospital, Ward 25, Oct. 1. 1873. Had a chill succeeded by high fever and pain in head and back. The following observations were made:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1873	M.	E.	M.	E.	M.	E.	
Oct. 20	78	74	28	26	104.5°	104.5°	Pain in head and back; nausea; tongue red at tip and edge; coated in center; conjunctiva congested; gums red and swollen; face flushed; capillaries of face, chest and extremities congested; high fever; intense thirst; passes very little urine and with great effort; iced milk and ice in mouth.
" 21	72	76	24	26	102°	103°	Bowels constipated; passes urine with great difficulty and experiences much pain in micturition. Pain in head, lumbar region and lower extremities, nausea and vomiting; tongue very red at tip and edges and coated in center; gums swollen and very red; mind heavy and confused; urine scant, albuminous. Ice water injections and sulpho-carb. of sodium.
" 22	66	72	22	24	101.1°	103°	Gums red, swollen and congested; tongue softer and not so red; urine contains albumen and casts; shows acid reaction; conjunctiva and surface yellow; nervous, restless. Ice water injections and sulpho-carb. of sodium
" 23	66	70	23	24	100°	100°	Improving; not so nervous; tongue clearing; urine albuminous, but more abundant. Continue sulpho-carb. of sodium, ordered also sulphate of quinia.

" 24	64	70	24	22	100.1°	101.1°	Continues to improve; urine albuminous with casts but more abundant; has some appetite; patient still very weak; continue quinia and sulpho carb. of sodium.
" 25	62	62	22	20	99.3°	101°	Improving. Sulpho-carb. of sodium continued; 20 grains every eight hours, as before.
" 26	64	60	23	23	99.1°	99.7°	Improving; continue sulpho carb. of sodium; tongue clearing off; skin clearing off; hungry.
" 27	60	59	22	18	97°	98.5°	Improving; although the urine is still loaded with albumen and casts; urine much more abundant and returns to normal standard; and sat up during the day.
" 28	60	56	18	20	97.6°	98°	Continues to improve; albumen disappears from urine.
" 29	60	76	18	24	98°	98.6°	Tongue clear and very near normal in appearance; urine abundant; is up and dressed.
" 30	60	62	18	24	98.8°	100.5°	Bowels regular; urine abundant; patient says that he feels well; walks about the ward.
" 31	60	60	18	28	99.1°	100°	Patient expresses himself as feeling well.
Nov. 1	68	68	20	20	99.5°	99.5°	Discharged in good condition November 10.

It is of interest to note that on October 22 the injections of ice-cold water were ordered and repeated three times. After the third injection, the patient passed quite a considerable amount of urine and fell into a refreshing sleep. The sulpho-carbolate of sodium, 10 grains every two hours, appeared to relieve the nausea. The patient slept well during the night and was able to drink a cup of milk and another of beef tea.

Case 7.—Yellow fever; severe case; jaundice; albumen in urine; recovery. Wm. Busse; age 25; native of Germany. Has been in New Orleans seven months, and came here from Red River. Seized with pain in the head and back and fever on Oct. 11, 1873, and entered Charity Hospital. Ward 24, October 13. At time of admission, complained of pain in head, back and knees. Tongue red at tip and edges and coated in the center. October 14, conjunctiva of eyes congested and yellow; surface of body yellow; nausea and vomiting; breath and odor of body foul; urine abundant and contains albumen and casts. On October 15 and 16, pulse intermittent, but the patient improving. On October 20, the patient was able to sit up and to be brought down to my clinical lecture in the amphitheater, and on October 22 was able to walk about the hospital. After the complete establishment of convalescence the patient was confined for several weeks by a painful abscess in the axilla. The following observations were made on the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 14	84	90	32	36	103.4°	103.8°
" 15	80	86	24	26	100.2°	102 °
" 16	82	84	26	24	100 °	100.2°
" 17	84	86	26	30	101 °	101.8°
" 18	82	78	26	24	99.6°	99.2°
" 19	80	82	22	24	99 °	100 °
" 20	84	84	26	24	98.6°	98 °
" 21	88	92	24	24	100 °	101 °
" 22	78	96	20	22	98.2°	99.8°
" 23	82	84	22	24	100.4°	100.4°
" 24	80	82	20	18	98.4°	99.3°
" 25	80	. . . .	20	. . . .	99.7°	. . . .
" 26	80	. . . .	26	. . . .	98.8°	. . . .

Case 8.—Yellow fever. Edward Ryan; age 42; native of Ireland; resident of United States twenty-five years, in New Orleans one year; laborer. Admitted to Charity Hospital, Ward 26. Oct. 8, 1873. Was sick since October 4. At time of entrance, high fever; red swollen gums; restless. On October 9 albumen and casts and bile appeared in the urine. On October 17, when the patient was convalescent, the urine contained but a small quantity of albumen. The following observations were begun on the sixth day of the disease and continued up to the nineteenth day and embraced the period of convalescence:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 11	75	70	31	36	100.7°	100.6°
" 12	68	72	30	36	98.6°	98.7°
" 13	60	61	34	36	98.6°	98.6°
" 14	60	66	36	30	98.9°	99.2°
" 15	58	56	32	32	99.2°	99.6°
" 16	58	60	26	26	99.4°	99.6°
" 17	52	62	30	36	99 4°	100.4°
" 18	60	58	30	30	100 °	100 °
" 19	58	60	28	26	99.3°	99.6°
" 20	60	62	28	28	98.2°	99.7°
" 21	80	80	28	28	99.7°	99.8°
" 22	80	70	28	26	99.2°	99.8°
" 23	86	68	26	28	100.3°	100 °

In the preceding case we observe that the lowest point in the action of the heart was reached on the seventeenth day of the disease, when the pulse was only 52, notwithstanding that the respiration was 30. It is also worthy of note that as in many other cases of yellow fever, both during convalescence and in the state of depression preceding death, the temperature fell below the normal standard. This, however, is not always so in fatal cases, which frequently terminate with high temperature.

Case 9.—Yellow fever followed by malarial fever. John Clooney; native of Liverpool; age 26. Entered Charity Hospital, Ward 26, Oct. 14, 1873. Patient had been working on rice plantation twenty-five miles above New Orleans. On October 1 was seized with chills and fever. Yellow fever appeared to have been engrafted on the paroxysmal fever. After producing its characteristic phenomenon, the malarial fever again appeared and was arrested by the free use of quinin.

Date.	Pulse.		Resp.		Temp.		REMARKS.
	M.	E.	M.	E.	M.	E.	
Oct. 14	100	118	20	28	100.8°	104.6°	Pain in head, epigastrium and back; perspiring freely; urine contains albumen, red color.
" 15	98	100	18	21	100.9°	101.5°	Perspiring: appetite gone; pains relieved; conjunctiva yellow and tongue slightly coated.
" 16	96	92	18	18	100.8°	100.4°	Improving: urine contains albumen
" 17	88	96	18	22	100.2°	100.5°	Improving; albumen in urine,
" 18	92	94	18	21	99.2°	100 °	Continues to improve; pulse intermittent.
" 19	80	84	18	24	99.8°	99.6°	Improving.
" 20	84	86	22	22	99.8°	102. °	Fever appeared in the day.
" 21	86	. .	22	. .	100.6°	. . .	Fever.
" 22	84	86	20	22	99.5°	102.2°	No albumen in urine; fever; pulse irregular and patient very nervous.
" 23	86	100	16	14	99.7°	102.6°	Fever, very nervous; hands tremble.
" 24	80	82	14	12	99.7°	100 °	Quinin freely administered; perspires; abatement of fever; patient very nervous; perspiring freely; respiration irregular and oppressed.
" 25	80	. .	14	. .	99.6°	. . .	Nervous; respiration irregular; twitching of muscles; no albumen in urine, and rested.
" 26	94	. .	16	. .	99.4°	. . .	Perspiration, rest and sleep during the night; hands and limbs very tremulous and nervous.
" 27	84	80	22	21	99.3°	100 °	Improving.
" 28	80	. .	18	. .	100.2°	100.2°	Improving.
" 29	80	84	18	18	100.2°	100.7°	Improving.
" 30	. .	. .	. .	. .	100.6°	. . .	

The diagnosis in this case presented some difficulty; with reference to the question whether the disease was yellow fever or remittent malarial fever. It is worthy of note that while the albumen was present in the urine of the yellow fever, it was absent during the subsequent attacks of malarial fever.

Case 10.—Yellow fever succeeded by intermittent fever; the latter disease arrested by quinin; recovery. H. M., age 22; native of Germany; butcher; has resided in New Orleans for four years. Says that he had fever four days before entering the hospital. Entered Charity Hospital, Ward 24, Oct. 3, 1873, with high fever, intense jaundice and capillary congestion. The fever intermittent October 8. The following record of temperature embraces the period of convalescence from yellow fever, and the appearance of the periodic malarial chills:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1873.	M.	E.	M.	E.	M.	E.	
Oct. 9	96	100	26	28	100.2°	100.6°	Intense jaundice; capillary congestion.
" 10	94	90	24	24	99.8°	100.4°	Intense jaundice; capillary congestion.
" 11	98	88	25	23	100.7°	100.4°	Intense jaundice: capillary congestion.
" 12	82	80	22	24	100.6°	100 °	Jaundice disappearing.
" 13	84	88	22	24	100.7°	100.8°	Improving; sitting up.
" 14	84	72	24	22	101.9°	99 °	Intermittent pulse.
" 15	82	104	22	25	100.6°	104.6°	Chill during day, followed by fever.
" 16	68	60	22	22	99.4°	99.7°	Intermission.
" 17	70	116	22	30	100 °	105 °	Chill at 3 p.m., followed by fever.
" 18	60	68	22	22	98.2°	99.4°	Intermission; quinin 5 grains four times every three hours.
" 19	78	78	26	22	100.8°	99.6°	Intermission; 5 grains quinin four times.
" 20	74	66	26	24	100.7°	99.8°	Intermission.
" 21	100	. .	26	. .	103 °	. . .	Chill during night.
" 22	*	*	*	*	*	*	Intermission; quinin freely given.
" 23	*	*	*	*	*	*	Intermission; quinin freely given.
" 24	*	*	*	*	*	*	Intermission; quinin freely given.
" 25	*	*	*	*	*	*	Intermission; quinin freely given.

* Normal.

In this case the patient had had repeated chills and fever during the summer and fall, which continued up to the time of the attack of yellow fever. This latter disease induced its cycle of changes, and was of a continuous type. Six days after the subsidence of the yellow fever febrile stage, the malarial fever reappeared at stated intervals and manifested its characteristic paroxysmal phenomena.

Case 11.—Yellow fever; hemorrhage from mouth and tongue; jaundice; pulse reduced during convalescence to 42 per minute; treated with sulpho-carbolate of sodium; recovery. Telespert Brutet; age 20; native of Canada; laborer. Has resided in New Orleans two weeks. Entered Charity Hospital, Ward 13, October 28. Attack of fever began with a chill which lasted three hours and was followed by intense headache and pain in epigastrium, back and lower extremities. From the account given by the patient, he appears to have entered the hospital on the fourth day of the attack. The following is the record of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.		Remarks.
1873.	M.	E.	M.	E.	M.	E.	
Oct. 28	*	*	. .	30	99.5°	102.2°	Conjunctiva yellow, face and chest with a tinge of yellow; tenderness of epigastrium; pain in head, back and lower extremities; nausea and vomiting; stomach irritable, retains nothing but fragments of ice. Vomited matter streaked with blood; tongue coated in the center, very red at tip and edges; gums red; mucous membrane of mouth red and ecchymosed in places; conjunctiva injected; surface injected; pulse feeble and irregular, cannot be counted; patient very weak; urine loaded with albumen and casts; strong acid reaction.
“	72	76	20	18	99 °	101 °	Thirst and dryness of mouth continue; jaundice increases; action of heart very feeble; urine red and albuminous.
“ 30	60	54	20	18	99.2°	99 °	Slept for a few hours; appears to be better; vomiting almost entirely arrested; pulse slower but stronger.
“ 31	44	44	18	16	97.6°	98.4°	Slept well during night; heart beats feeble; respiration slow but deep drawn; improving; gums and tongue bleeding; spits blood; urine albuminous.
Nov. 1	42	50	14	16	98.2°	101.8°	Hemorrhage from tongue and gums continues; heart actions feeble; urine albuminous; general symptoms improving; jaundice well marked.
“ 2	50	54	20	23	98.2°	101.8°	Heart still beating feebly: hemorrhage from tongue and gums; otherwise improving.
“ 3	54	44	18	18	98.6°	98 °	Tongue moist and not so red; hemorrhage stopped; action of heart still very feeble.
“ 4	44	. .	17	. .	98.6°	. . .	Improving.
“ 5	44	. .	16	. .	98.8°	. . .	Improving.
“ 6	46	42	20	20	99.4°	99.8°	Continues to improve.
“ 7	48	. .	20	. .	98 °	. . .	Convalescent.

*Feeble. irregular.

This patient was treated from October 28 to November 2 with 20 grains of the sulpho-carb. of sodium every three hours.

In the preceding case, coupled with a small amount of black vomit and continuous hemorrhage from the mucous membrane of the mouth, there was depressed temperature, slow pulse and a very feeble action of the heart. Although the prognosis in this case was unfavorable for at least six days, the patient passed by slow stages to complete recovery.

The following cases of yellow fever treated in my wards of the Charity Hospital in the autumn of 1873, were all attended to a greater or less extent with jaundice and the observations upon the pulse, respiration and temperature relate to the period of convalescence:

Case 1—E. S., age 27; attacked with yellow fever September 18. During convalescence, while the conjunctiva and skin were still yellow, the pulse ranged from 56 to 61, respiration from 17 to 22, and the temperature between 98.5 and 100 degrees.

Case 2.—J. J., age 21; laborer; native of Wales; contracted fever in Memphis, Tenn., September 10; during convalescence while still jaundiced, pulse 68 to 97; respiration 22 to 28; temperature 99.5 to 102 degrees.

Case 3.—H. S., age 20; native of Germany; laborer; attacked Sept. 29, 1873. During convalescence, pulse 43 to 70; respiration 12 to 14; temperature 99 to 100 degrees.

Case 4.—H. A., age 40; native of Germany; attacked with pain in head and back, and fever Oct. 3, 1873; entered Charity Hospital, Ward 27, October 6. October 8, evening, pulse 79; respiration 28; temperature 102 degrees. October 9, morning, pulse 92; respiration 27; temperature 102.5 degrees; evening, pulse 94; respiration 27; temperature 102.5 degrees. October 10, morning, pulse 78; respiration 22; temperature 97.9 degrees; evening, pulse 75; respiration 22; temperature 99 degrees. October 11, morning, pulse 58; respiration 19; temperature 97 degrees; evening, pulse 63; respiration 21; temperature 98.7 degrees. October 12, morning, pulse 66; respiration 20; temperature 98; evening, pulse 56; respiration 20; temperature 98 degrees. October 13, pulse 64; temperature 97.5 degrees.

Case 5.—F. H., age 18; native of Kentucky; laborer; admitted to Charity Hospital, Ward 13, Oct. 3, 1873, second day of disease. October 8, convalescent, but still jaundiced; pulse 50; respiration 20; temperature 98.5 degrees. October 9, morning, pulse 54; respiration 20; temperature 97.4 degrees; evening, pulse 62; respiration 20; temperature 98.6 degrees. October 10, morning, pulse 56; respiration 18; temperature 97.8; evening, pulse 58; respiration 20; temperature 99.6. Discharged October 12.

Case 6.—G. O., age 35; native of Ireland; laborer. Entered

Charity Hospital, Ward 25, on third day of disease. October 8, moderate fever; pain in head, back and epigastrium; evening pulse 93; respiration 23; temperature 101 degrees. October 9, morning, pulse 89; respiration 29; temperature 100 degrees. Conjunctiva and skin yellow; urine contains bile and albumen; evening, pulse 85; respiration 26; temperature 100 degrees. October 10, morning, pulse 85; respiration 23; temperature 99.5 degrees; evening, pulse 93; respiration 26; temperature 99.5 degrees. On the 9th the urine was suppressed; great capillary congestion of the extremities, face and neck; black vomit during the night of the 10th

Post-mortem examination revealed the characteristic lesions of yellow fever. If the last case be compared with the four preceding cases, which terminated favorably, we observe that in them the pulse was not so accelerated as in the fatal case, with urinary suppression and black vomit.

Case 7.—Yellow fever; jaundice; recovered. L. K., age 19; native of France; taken sick on October 18, 1873 with a chill followed by fever which continued for five days, up to 8 P.M., on October 23. During the period of convalescence, extending from the fifth to the twelfth day of the disease, the temperature ranged from 102 on the fifth day to 97.5 degrees on the eighth day; the pulse ranged from 60 to 86 beats per minute, and the respiration from 20 to 30 per minute. The pulse descended gradually from 86 on the fifth day with a temperature of 102 degrees, to 60, with a temperature of 98.8 degrees, on the tenth day of the disease.

Case 8.—Yellow fever. Maurice Pierre; age 28; native of Switzerland; came down the Mississippi River from St. Louis, and in passing Memphis went ashore for a short time. After having been in New Orleans for five days, the patient was seized with a chill on October 19, which was followed by a fever of continued type and which did not abate until October 22. Entered the Charity Hospital evening of October 21 with a temperature of 103.5 degrees. From the fourth to the ninth day of the disease, the temperature ranged from 97.5 to 101.2 degrees; pulse from 56 to 78; respiration from 24 to 32. Both the respiration and the pulse were at times intermittent. The urine contained but small quantities of albumen and the jaundice was comparatively slight in this case; notwithstanding the apparent mildness of the fever, the pulse descended to 56 beats per minute and was intermittent and irregular.

Such facts indicate that the slow and intermittent action of the heart in yellow fever during convalescence and in fatal cases during the period of calm or depression following the active febrile stage, is not solely due to such matters as the bile in the blood, but rather to the structural alterations of the muscular fibers which I have fully described.

Case 9.—Yellow fever; jaundice; slow pulse; recovery. Dennis Manning; age 28; native of Ireland. Has resided in New Orleans seven years. Entered Charity Hospital, Ward 24, November 4. Tongue red at tip and edges; gums congested, red and bleeding; intense thirst; vomiting; can retain nothing upon stomach; great capillary congestion; restless and sleepless; hiccough; urine loaded with albumen and casts. Ordered sulpho-carbolate of sodium, 20 grains every four hours; ice bag to epigastrium; ice-water injections to rectum; iced milk; fragments of ice in mouth.

November 7, pulse 66; respiration 20; temperature 100 degrees at 8 A.M.; November 8, at 8 P.M. 101 degrees; conjunctiva yellow and congested; surface of body yellow and congested; 8 A.M., pulse 60; respiration 20; temperature 98.5 degrees. At 8 P.M., temperature 99. Restless and prostrated; urine loaded with albumen and casts.

November 9, 8 A.M., pulse 66; respiration 16; temperature 99 degrees; 8 P.M., pulse 66; respiration 14; temperature 98 degrees. Tongue red at tip and furred, cracked and bleeding; gums congested and bleeding; great muscular prostration; action of heart slow—beats feeble. Treatment continued.

November 10, 8 A.M., pulse 60; respiration 20; temperature 98 degrees. 8 P.M., pulse 60; respiration 20; axilla 99 degrees.

November 11, tongue and gums red and bleeding; jaundice and great congestion of capillaries of surface and extremities which present a purplish mottled appearance; urine loaded with albumen; dizziness in head. At 8 A.M., pulse 52; respiration 20; temperature of axilla 97.6 degrees. At 8 P.M., pulse 68; respiration 20; temperature 100. Improving; bleeding from tongue and gums almost entirely arrested; dizziness in head still continues; pulse slow; but action of heart is feeble and patient incapable of any great exertion.

November 12, pulse 52; respiration 18; temperature 99.2 degrees. At 8 P.M., pulse 54; respiration 20; temperature 100 degrees.

November 13, 8 A.M., pulse 51; respiration 20; temperature 99 degrees. At 8 P.M., pulse 48; respiration 20; temperature 99 degrees.

November 14, 8 A.M., pulse 48; respiration 16; temperature 99.5 degrees. Continued to improve and was discharged in good health.

Case 10.—Yellow fever, engrafted on paroxysmal malarial fever and followed by remittent fever and phthisis pulmonalis. Charles Scott; age 32: native of New Brunswick;

carpenter; resided in New Orleans for eight months. Admitted to Charity Hospital, Ward 13, Sept. 29, 1873. Says that three days before entering the hospital he was seized with a chill which lasted five minutes, and was followed by a fever of eight hours duration. The fever was again followed by chill and fever. After the relief of the paroxysmal fever by sulphate of quinia, a continuous fever with pain in head and back appeared on October 5. The following observations were recorded in this case, illustrating the superinvention of paroxysmal fever during convalescence:

Date.	Pulse.		Resp.		Temp.		REMARKS.
1875	M.	E.	M.	E.	M.	E.	
Oct. 8	. .	67	. .	25	. . .	100 °	Patient has no fever but has pains in lumbar region.
“ 9	60	64	22	22	99.4°	100 °	Feels fairly well, but weak; albumen in urine.
“ 10	60	62	20	22	99.6°	101.3°	Appears to be improving; albumen in urine.
“ 11	52	59	25	24	100.4°	101.2°	Did not rest well during night: tongue furred and very dry: thirsty; drank ice water but vomited it up.
“ 12	52	52	22	22	100.2°	101 °	Conjunctiva and skin yellow; swollen; tongue red and apparently with spots of blood on it.
“ 13	51	56	24	28	99.5°	100.5°	Jaundice; heaviness; red gums and tongue; pain and heaviness in epigastrium.
“ 14	50	58	26	26	100.1°	100.5°	Severe pains in epigastrium; tongue red at tip and edges; gums red; jaundice.
“ 15	52	62	26	28	100 °	101.5°	Jaundice; urine abundant, albuminous.
“ 16	54	60	30	28	100 °	101.5°	Jaundice; urine abundant, albuminous; pain in epigastrium and lower extremities.
“ 17	48	58	28	26	100.5°	102.5°	Jaundice and great congestion of capillaries; urine abundant and contains albumen and casts; pains in epigastrium and lower extremities.
“ 18	54	52	30	30	101.2°	101.2°	Congestion of capillaries; jaundice.
“ 19	50	52	24	18	101 °	102 °	Fever in evening; patient very weak, cannot sit up in bed; nervous; pains in epigastrium.
“ 20	70	56	28	26	101 °	102 °	Feeling of uneasiness in epigastrium; albumen in urine; jaundice diminishing.
“ 21	72	80	28	28	101.1°	102.2°	Jaundice diminishing; patient feels stronger.
“ 22	78	80	26	26	101 °	102 °	Still very weak and exhausted by least motion.
“ 23	58	58	20	20	100.5°	101 °	Appears to be much better.
“ 24	. .	. .	. .	. .	103 °	103 °	Paroxysm of fever.
“ 25	. .	. .	. .	. .	103 °	101 °	Fever; quinin freely administered.
“ 26	74	76	32	24	101 °	98 °	Fever subsided.
“ 27	80	68	24	28	99 °	101.5°	Convalescent.
“ 28	80	. .	26	. .	101 °	. . .	Convalescent.

After the subsidence of the yellow fever it was found that the temperature did not subside, but still kept up, and upon careful examination I found evidence of phthisis pulmonalis in both lungs. The following is the record of the pulse, temperature and respiration after the subsidence of the yellow fever:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 28	. . . .	72	. . . .	28	102.5°	103°
" 29	88	88	24	32	102°	103°
" 30	88	94	28	30	101.5°	103.5°
" 31	84	. . . .	28	. . . .	101.5°	103.5°
Nov. 1	92	. . . .	28	. . . .	101°	103.5°
" 2	92	. . . .	32	. . . .	101°	103°
" 3	90	88	24	36	101.5°	103°
" 4	90	. . . .	24	. . . .	100°	103°
" 5	94	88	23	24	100.8°	101.2°
" 6	84	79	24	24	100.6°	101.5°
" 7	. . . .	100	. . . .	32	101°	103.2°
" 8	80	. . . .	28	. . . .	100.5°	101.6°
" 9	84	88	32	28	101.5°	102.5°
" 10	84	88	28	30	101.5°	102.3°
" 11	. . . .	90	. . . .	28	101.5°	103°
" 12	. . . .	100	. . . .	30	101.5°	103°
" 13	88	88	26	32	101.2°	101.5°
" 14	90	84	28	24	102.2°	102.5°
" 15	98	64	28	28	102°	101°
" 16	90	. . . .	36	. . . .	102°	. . . .

Case 11.—Yellow fever. Charity Hospital, Ward 25; John Pflum; age 26; native of St. Louis; fever begun with a chill of half an hour's duration, followed by fever on October 7. The fever continued up to the time the patient entered the hospital, Oct. 20, 1873. Had been sick two days before entering the hospital.

Date.	Pulse.		Resp.		Temp.	
1873.	M.	E.	M.	E.	M.	E.
Oct. 20	78	74	28	26	104.5°	104.5°
" 21	72	76	24	26	102°	103°
" 22	66	72	22	24	101.1°	103°
" 23	66	70	22	24	100°	100.2°
" 24	64	70	24	22	100.1°	101.1°
" 25	62	62	22	20	99.3°	100°
" 26	64	60	23	25	99.1°	99.1°
" 27	60	59	22	18	97°	98.5°
" 28	60	56	18	20	97.6°	98°
" 29	60	76	18	24	98°	98.6°
" 30	62	. .	18	. .	98.2°	. . .

REMARKS.

October 20.—High fever; skin congested; conjunctiva of eyes very much congested and injected; tongue furred and red at edges; great pain in forehead and throat; passes urine only with great effort; ordered iced milk and ice fragments in mouth; gums congested.

October 21.—Passes urine with great effort; urine loaded with albumen; complete suppression of urine during night; grows restless and talks at random during the night.

October 22.—Ice cold injections ordered and repeated three times; after the third injection, passes quite a quantity of urine; when patient gets to sleep complains of nausea, when the sulpho carb. of sodium was ordered in 10 grain doses, repeated every two hours, which afforded relief to the symptoms and nausea.

October 23.—Patient slept well during night; has been able to drink a glass of milk and cup of beef tea; during the night, passes urine quite freely, loaded with albumen, and urine is greenish-yellow in color.

October 24.—Bowels moved; feels great relief and expresses himself as better; continue the sulpho carb. of sodium as above ordered; drinks milk and beef tea; urine passes pretty freely; albumen present in urine but decreasing in amount; complains of hunger; bowels moved.

October 26.—Tongue clearing off; skin begins to clear up; great hunger; urine returning to the normal standard; quantity of urine increasing.

October 27.—Patient sat up during the day.

October 28.—Goes about the ward, but watched; albumen disappears from urine.

October 29 and 30.—Patient goes about, is discharged and goes out cured.

Case 11.—Yellow fever. Adolph Kammer; age 21; native of Sweden, has been in America six years. in St. Louis, Mo. Admitted in the Charity Hospital, Ward 13, November 1, 8 P.M. Had been in New Orleans two weeks; sick two days previous to entrance.

Date,	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Nov. 2	84	82	32	43	104.5°	105 °
" 3	86	96	32	34	102 °	102.5°
" 4	72	70	28	36	101.5°	102 °
" 5	54	48	26	24	98.5°	101 °
" 6	52	50	26	22	98.3°	98 °
" 7	46	50	22	24	97.5°	98.5°
" 8	45	54	22	22	97.7°	98 °
" 9	52	50	26	27	97.8°	98 °
" 10	48	48	20	19	98 °	98.2°
" 11	52	52	26	24	98 °	98.2°
" 12	54	52	24	24	98 °	98.2°
" 13	52	46	26	24	98 °	98.2°

REMARKS.

November 2.—Skin hot and dry; slight jaundice; gums congested, tongue dry and swollen; kidneys acting imperfectly. Passed a small quantity of urine by catheter; urine loaded with albumen; ice water injection caused passage of urine during day and evening.

November 3.—Complains of stomach, but relieved by ice sprays to epigastrium. Has been treated with sulpho carb. of sodium, ʒij divided into 6 powders, one powder (20 grains) every four or six hours; milk punch, brandy and beef tea, and small lumps of ice in mouth. At night slight hemorrhage from the gums; passed urine quite freely towards the morning of November 4; urine dark colored and loaded with albumen and bile.

November 4.—Very quiet during the day; perspires freely and seems to be in better condition; passes urine freely. Continue sulpho carb. of sodium in the same doses. Milk punch, beef tea and ice. Wash the face and hands with tincture of camphor and water; this seems to refresh him greatly. Bowels have been moved.

November 5.—Passed a good night, had some sleep, begins to look more cheerful and desires to sit up.

November 6.—Passed a very comfortable night; jaundice disappearing; urine freely passed; urine still contains albumen, but the quantity is diminished.

November 7.—Doing well, sits up; all pain disappeared, although quite weak; eats and sleeps well; jaundice disappearing.

November 8.—Patient still improves; albumen disappeared from urine; walks around.

November 9, 10, 11 and 12.—Rapidly improves; jaundice disappearing rapidly; slight traces of yellow in eyes.

November 13.—Discharged from observation and leaves, November 17, quite well.

Case 12.—Yellow fever. Wm. Thompson; age 23; native of Germany has been in Louisiana eighteen months. Taken sick in the country with intermittent fever; returned to the city after a few days; was seized with a violent chill, followed by continued fever until day of entrance into the hospital. Large muscular man; blacksmith by trade. Entered Charity Hospital October 27. Patient had been in bed twenty-four hours with fever before entering hospital.

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 27	....	104	....	28	....	104.2°
" 28	88	98	28	29	104°	105°
" 29	86	88	26	26	103°	105°
" 30	98	96	39	22	103°	104°
" 31	80	86	28	30	102°	102.5°
Nov. 1	82	86	36	32	102.5°	102.8°
" 2	82	94	34	40	102°	104°
" 3	92	99	43	38	103.2°	103°
" 4	90	94	34	40	100.5°	101°
" 5	106	104	40	50	102.5°	102°
" 6	106	118	36	38	102.5°	103.8°
" 7	126	129	35	56	100°	104.2°

Remarks.

October 27.—Was much excited: face flushed; gums red; tongue furred: eyes injected, hemorrhage from gums; some headache: cannot bear the gas light; great thirst; ice in small fragments, milk and brandy diluted with seltzer water.

October 28.—Did not sleep during night; relieved the bladder by the catheter: urine scant and high colored, loaded with albumen; ordered sulpho carb. of sodium ʒij in 6 powders, one powder every three or four hours.

October 29.—Did not sleep during night: restless; skin dry; great pain in region of kidneys; no urine is passed; ice cold injections caused the excretion of a very small quantity of light-colored urine loaded with albumen. Hemorrhage from the gums profuse, also free epistaxis; patient greatly alarmed and restless.

October 30.—No improvement: takes little milk and seltzer water and brandy; skin is still dry; tongue dry and bleeding.

October 31.—Patient has not slept at all and is in a state of low delirium; passes his urine and feces in bed; lies in an apparently insensible condition, but taking nourishment in small quantities when forced upon him.

November 1.—No change, except for the worse; still in an unconcious state; low muttering delirium; cannot collect any urine; passes the small amounts in bed; severe hemorrhage from stomach and lungs; expectorates blood.

November 2.—No change; in same condition.

November 3.—Loud stertorous breathing; hemorrhage; blood still runs out of his mouth.

November 4.—Continues to grow worse; body emits a horrible stench; expectorates blood; blood oozes from nostrils and gums

November 5.—Wild delirium, requiring two men to hold him in bed.

November 6.—Continues in state of wild delirium.

November 7.—Sinking rapidly; died 1 a.m., morning of Nov. 8, 1873, with profuse hemorrhage.

Post-mortem Examination.—Eight hours after death. Body of a deep golden color; mouth full of black vomit, which trickled down the side of the body; bladder contained not more than ʒij of light-colored urine; stomach was filled with black vomit, and capillaries were congested; kidneys congested and mottled; liver of a nutmeg color and oily when first seen, when washed and exposed to the air sections of the liver presented a true yellow color; had yellow flabby and fatty old adhesion in right lung; blood thin and watery. All organs showed capillary congestion. Gall bladder contained but a small quantity of bile.

Case 1.—Yellow fever, complicated with pneumonia. Wm. Thompson, large muscular man, blacksmith by trade; age 22; native of Germany. Entered Charity Hospital, Ward 13, Oct. 27, 1873. Has resided in Louisiana eighteen months. Went out to Bayou Têche to work on plantation; after remaining there two weeks, returned to the city, and after being in New Orleans one week, was taken with a slight chill, followed by fever October 27, the day upon which he entered the hospital. The following is the record of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 27	. . . .	104	. . . .	28	. . . .	105.5°
" 28	88	98	28	29	104°	105°
" 29	86	88	26	26	103°	105°
" 30	98	96	39	22	103.5°	104°
" 31	80	86	28	30	102°	102.5°
Nov. 1	82	86	36	32	102.5°	102.8°
" 2	82	94	34	40	102°	104°
" 3	92	99	43	38	103.2°	103°
" 4	90	94	34	40	100.5°	101°
" 5	106	104	40	50	102.5°	102°
" 6	106	118	36	38	102.5°	103°
" 7	126	121	35	56	100°	104.2°

REMARKS.

October 27.—Very much excited; face flushed; eyes injected; gums red; cannot bear gas light; hemorrhage from gums; entered evening of October 27; pain in head and back; hot skin; restless; anemia.

October 28.—Tongue very red at tip and edges; bled from nose at 1 p. m. and 7.30 p. m.; complains of some pain in forehead, lumbar and epigastric regions and extreme capillary congestion; foul breath and odor.

October 29.—Complains of intense headache and sleeplessness; epigastrium tender upon pressure and painful; great pain in region of kidneys; suppression of urine.

October 30.—Albumen and casts in urine; jaundice; capillary congestion; did not sleep during night; hemorrhage from gums.

October 31.—Jaundice; capillary congestion; prostration; suppression of urine; ice cold injections cause the passing of some urine loaded with albumen.

November 1.—Intense jaundice.

November 2.—Profuse bleeding at nose; blood oozes from tongue and nose; dullness upon percussion of lower lobes, both lungs; embarrassed respiration.

November 3.—Bloody sputa; blood oozes from gums.

November 4.—Bloody sputa and bleeding gums.

November 5.—Bloody sputa; blood oozes from tongue and gums and nose.

November 6.—Bloody sputa; blood oozes from mouth and nose.

November 7.—Bloody sputa; died at 12 p. m.

NECROPSY TEN HOURS AFTER DEATH.

Body still warm; limbs, face and trunk full; surface of deep saffron yellow color; intense jaundice; lower dependent portions of head, neck, trunk and extremities mottled with dark purplish ecchymoses. Black vomit running out of corners of mouth. Thorax: Heart firmer and more contracted and of not so decided a yellow color as in uncomplicated yellow fever. Under the microscope, however, the muscular fibers were loaded with granular matter and oil globules. The pericardium contained two fluid ounces of yellow serous fluid. The right auricle contained a golden-colored clot, attached to the auriculo-ventricular valve. The formation of the clot appeared to be due to the supervention of the pneumonic inflammation. The lungs were congested; lower lobes of both lungs solidified, in state of gray hepatization and infiltrated with bloody purulent fluid. In the blood from the cavities of the heart, the majority of the colored blood corpuscles were normal in appearance; some were crenated and stellate in shape. No bacteria or vegetable or animal organisms were observed in the blood from the heart. Abdomen: Liver mottled upon surface, yellow and purple and bronze. Gives evidence of pervading malarial disease. The yellow fever deposit of oil globules and yellow granular matter was very evident in many portions of the liver. Under the microscope the liver was loaded with oil globules and granular matter and also contained black pigmentary matter. Gall bladder relaxed and apparently empty, contained about thirty grains of thick deep green, almost black, ropy liquid, which under the microscope was found to consist of desquamated liver cells, granular casts of the biliary tubes, and cells from the walls of the gall bladder, discolored by the coloring matter of the bile. The bile in cases complicated with malarial fever, presents a deep green color. Walls of gall bladder greatly congested and of deep purple color. Spleen enlarged to about twice the size and contained pigment particles. Kidneys congested; when washed, of deep yellow color. The bladder contained small quantity of golden urine, loaded with albumen casts and cells of tubuli uriniferi. Stomach and intestinal canal congested. Mucous membrane of stomach deeply congested.

Case 2.—Yellow fever; jaundice; severe pain in heart; slow pulse; death. James H. Campbell; age 48; native of New York; has resided in New Orleans twenty-two years; occupation, carpenter. Seized with pain in head, followed by fever Oct. 17, 1893. Entered Charity Hospital October 21.

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 21	. . . .	. . . .	. . . .	28	. . . .	103.5°
" 22	90	94	26	30	102 °	103.5°
" 23	80	80	34	30	100.1°	100 °
" 24	77	78	30	26	99.2°	100.1°
" 25	80	82	28	27	101.7°	103 °
" 26	95	100	28	30	101 °	104 °
" 27	. . . .	. . . .	. . . .	. . . .	101 °	. . . .
" 28	60	56	42	40	99 °	99 °

REMARKS.

October 21.—Conjunctiva and skin very yellow; tongue red at tip and edges, coated and deeply furred in center; gums very red; patient emits a highly offensive odor.

October 22.—Jaundice, nausea and vomiting; bowels moist; very offensive smell emitted by patient; respiration rapid and panting; dull and drowsy.

October 23.—Patient complains of intense pain in the region of the heart, expressive of fright; in the evening partial suppression of urine; ice cold water administered by enema.

October 24.—During this and the preceding day the tongue and mouth have been so dry that the tongue is protruded with great difficulty: tongue furred and cracked; very red at tip and edges; intense jaundice; tongue swollen; speaks with great difficulty, dull and lethargic; urine contains albumen, granular casts and detached cells. Ice water injections into rectum has caused free secretion of urine.

October 25.—Tongue swollen, cracked and bleeding; jaundice intense; upper surface of tongue red and furred, under surface purple.

October 26.—There has been a marked rising temperature.

October 27—Complains of intense pain in right hypochondriac region; countenance anxious and disturbed; jaundice.

October 28.—Pulse slow, only 56 per minute.

Died Oct. 28, 1873, two or three hours after the last observation. Died with slow pulse, low temperature and rapid respiration. This patient entered in a state of intense jaundice and great prostration. Pulse so slow that it could not be counted.

Case 3.—Yellow fever; jaundice; urine contained albumen; rapid pulse; death. A. L. W.; native of Ireland; age 20; sailor; in port one month. Taken sick October 22, and was sent to the Charity Hospital three hours after beginning of illness. October 22, 1874, 8 P.M., pulse 125, respiration 35; temperature of axilla 104.2 degrees. October 23, urine contains albumen and casts; congestion of capillaries; face very much congested; tongue red at tip and edges, furred in center; gums swollen and very red; great tenderness of epigastrium on pressure; 8 P.M., pulse 118; respiration 30; temperature 102.2 degrees; 8 P.M., pulse 120; respiration 32; temperature 104.1; urine loaded with albumen and casts; very scant. October 24, nausea and vomiting black vomit; can retain nothing on his stomach; 8 A.M., pulse 105; respiration 26; temperature 101.5 degrees; 8 P.M., pulse 102; respiration 25; temperature 102.4 degrees; urine scant, could collect but one fluid ounce; loaded with albumen and casts; black vomit continues. October 25, 8 A.M., pulse 96; respiration 24; temperature 100.2 degrees; 8 P.M., pulse 107; respiration 26; temperature 99.4 degrees; black vomit; urinary suppression; died shortly after last observation, October 26, 4 A.M. This case, which ran a rapid course, terminated with a temperature of the axilla but little elevated above the normal standard. The diminution of the rapidity of the action of the pulse and respiration, also corresponded with the diminution of temperature.

Case 4.—Yellow fever; jaundice; formation of bed sores and large abscess on thigh; death at the end of four months from effects of abscess of thigh; the pulse during convalescence was more rapid than in uncomplicated cases of yellow fever, which pass to convalescence and complete recovery, without the supervention of local inflammations. Emanuel Scheise; age 37; native of Germany. Has been residing on Frenchman Street. Entered Charity Hospital, Ward 25, Oct. 29, 1873. On entering the hospital the patient stated that he had been suffering with a high fever for three days. This fever was accompanied with violent pains in supraorbital, lumbar, and epigastric regions, and pains in the eyes and lower extremities. At the time of his entrance into the hospital the patient was jaundiced, had fever, nausea, and hemorrhage from the nose and gums. The capillary circulation was sluggish and the urine scant, albuminous, with casts of the tubuli uriniferi. The patient was treated with the sulpho-carbolate of sodium, 20 grains every four hours; ice water injections to the rectum, brandy and water charged with carbonic acid gas, iced milk, beef tea and ice bags to epigastrium. Although hemorrhage took place from nose and gums and jaundice supervened the patient passed safely through the active stages of yellow fever. When, however, convalescence should have been established, the pulse and temperature continued above the normal standard; the patient complained of great pain in left groin and thigh. The limb became enormously swollen. A large abscess finally formed which was opened on January 7 and discharged over half a gallon of offensive pus. The patient died February 24, after prolonged and intense suffering from the abscess and from several large bed sores, on the hips and over the sacrum. The following is a record of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Oct. 30	100	98	18	18	103.6°	103 °
" 31	88	92	20	20	100.6°	101.6°
Nov. 1	82	82	18	18	100.4°	101 °
" 2	78	88	20	20	100.6°	101.8°
" 3	100	98	21	24	103.8°	103.7°
" 4	100	....	22	....	102.6°	....
" 5	100	....	22	....	102 °	....
" 6	96	96	22	24	102 °	102.6°
" 7	86	84	20	20	102.4°	101 °
" 8	74	90	18	22	101.2°	102 °
" 9	70	80	20	22	100.6°	100.8°
" 10	82	....	20	24	100.4°	101.5°
" 11	70	110	20	24	99.7°	101 °
" 12	72	98	18	18	99 °	101.2°
" 13	85	98	20	22	99.2°	101.8°
" 14	100	98	18	18	100.8°	101.4°
" 15	94	....	20	....	100.4°	....
Dec.	90	110	20	24	100 °	103 °
Jan.	100	120	24	28	101 °	104 °
Feb.	110	140	28	30	101 °	104 °

REMARKS.

October 30.—Complains of great weakness; conjunctiva of eyes yellow; surface slightly yellow; tongue very red at tip and edges; gums red and swollen; teeth covered with sordes; breath and odor of body very offensive; bowels constipated; passes urine freely; stomach irritable; tenderness of epigastrium; hemorrhage from gums and nose; thirsty, great dryness of mouth and throat.

October 31.—Urine contains albumen and yellow granular casts, and cells from tubuli uriniferi; eyes congested, surface congested and yellow; pain in epigastrium, nausea relieved; conjunctiva of eyes yellow and congested; tongue red at tip and edges, and heavily coated in the center; gums red and swollen; urine high colored.

November 1.—Diarrhea; stools are very offensive and of a light yellow color; jaundice, capillary congestion; eyes congested, red and jaundiced; complains of pain in right ear.

November 2.—Complains of severe pains in groins; urine contains albumen casts and bile.

November 3.—Pulse frequent, full and strong; pains in groins very severe; lymphatics swollen and painful.

November 4.—Pulse frequent and strong; has fever; pain located in left thigh, which is swollen, painful and tender; albumen in urine, but diminished.

November 5.—Pain and swelling in thigh continue.

November 6.—Perspiring freely; pain in thigh very severe, it is necessary to use opium freely; the bowels are kept open and the patient supported with tincture of cinchona.

November 7.—Thigh swollen and hard and very painful; thigh painted with iodin, tincture of bark and sulpho-carb; alcoholic stimulants.

November 8.—Pain and swelling of thigh; applied ointment, compound of tincture of iodin and opium, f. ʒij and sulpho-carb. ʒij.

November 9.—Opium internally; with bitter tonics and alcoholic stimulants applied, together with the local applications to be productive of good.

November 10.—Pain less; improving; albumen in very small amount.

November 11.—Pain less; appears to be improving.

November 12.—Thigh not so painful, but still much swollen; patient sits up with some difficulty.

November 13.—Weak, but with some appetite.

November 14.—Pain and swelling in thigh continues; whole thigh from hip to knee swollen.

November 15.—Pain and swelling in thigh not so intense.

December.—During the remainder of November and during December swelling of leg continues, also hectic fever; temperature rises from 100 to 103 degrees; pulse 90 to 110.

January.—On the 7th of January abscess of thigh opened, discharging a large quantity of fetid matter; pulse report 100–120; temperature 101 to 104 degrees.

February.—During the months of February and January had sores formed upon hips and on sacrum; hectic fever; loose bowels; rapid pulse; great emaciation; fever; temperature from 101–104 degrees. Died worn out by fever and suffering Feb. 24, 1873.

In this case the patient passed safely through a severe attack of yellow fever, and then fell a victim to one of the occasional results of this disease, namely, abscess. The effects of the local inflammation in presenting the marked depression of the pulse are clearly shown in this case. I have recorded many cases illustrating the marked reduction of the beats of the pulse during the stage of so-called calm or depression and of convalescence in yellow fever, and will only adduce the following:

Case 5.—Yellow fever; jaundice; black vomit; hemorrhage from bowels; urinary suppression; fatal issue. Jacob Geir, native of Germany; age 39 years. Several years in this country. During the past seven months has been in the South, on a plantation near Baton Rouge. The white laborers, as well as himself were all attacked with fever. About two months before entering Charity Hospital, came to New Orleans, and was at that time suffering with malarial fever, which recurred at intervals up to the time of his admission. Admitted to Charity Hospital, Ward 22, Nov. 22, 1873, in the evening. November 23, 8 A.M., conjunctiva of eyes congested and yellow; skin of face and trunk yellow and congested; tongue red at tip and edges and furred in center. When the fingers are pressed upon the surface white spots remain into which the blood slowly returns and restores the florid congested appearance due to sluggish capillary circulation. Pulse 96; temperature 103.5 degrees. November 24, 8 A.M., pulse 84; temperature 101.2 degrees. Bowels loose during the night. Urine scant; nausea; threw up black vomit at 9:30 A.M. Reaction of black vomit strongly acid; urine albuminous, with granular casts of urinary tubes and desquamated cells from excretory tubes. ℞. Sulpho-carbolate of sodium ʒ iij. Divide into six powders. One powder every six hours. 8 P.M., vomiting of black vomit continues. Since this has begun the temperature has progressively declined. Pulse 82; respiration 30; temperature of axilla 98 degrees. November 25, during the night passed by the bowels black excrements mixed with blood. Only three fluid ounces of urine have passed during the last twenty-four hours. Urine yellow, turbid, loaded with albumen, casts and cells. Casts and cells of deep yellow color. Great capillary congestion; dependent portions of body mottled and of a purplish yellow hue. Hands and feet cold; the depression of temperature extends up to elbows and knees. Pulse can be scarcely felt at wrists. Temperature 98 degrees; pulse 70; respiration 26. 6 P.M., temperature 100 degrees; pulse 60; respiration 24. Black vomit and bloody black discharges from bowels still continues. November 26, 8 A.M., insensible; pulseless; temperature 98 degrees; respiration 20. Died at 11:30 A.M.

Case 6.—Yellow fever; black vomit; death. *Post-mortem* examination three hours after death; characteristic lesions of yellow fever. Daniel McMenagale; age 18; native of Ohio; left his native State two years previous to the present attack; resided in Memphis, Granada and Vicksburg; came from the latter place, and has resided in New Orleans two weeks. Has had chills and fever at various times during the past two months. Entered Charity Hospital Oct. 11, 1873, 6 P. M. I saw this patient for the first time at 8 A.M., October 12. Intellect dull; patient unable to give a connected account of the present attack of fever. Surface of body covered with petechiæ; gums red; tongue red at tip and edges; body of tongue coated with yellow fur. Epigastrium tender on pressure. Conjunctiva of eyes yellow and injected.

Tongue cracked and bloody. Blood oozes from the tongue, and also from the gums. During the night the patient vomited freely, and the matters ejected from the stomach contained flakes of dark blood. Only a small quantity of urine, about two fluid ounces were passed, which contained albumen and bile, and casts of the tubuli uriniferi and excretory cells. Injections into the rectum of ice-cold water were ordered, and these appeared to restore to a limited extent the action of the kidneys: The following table embodies the prominent symptoms:

Date.	Pulse.		Resp.		Temp	
1873	M.	E.	M.	E.	M.	E.
Oct. 12	110	112	22	26	104.6°	105.5°
" 13	110	112	29	34	104.5°	104.7°
" 14	116	130	35	38	103.3°	103.3°

REMARKS.

October 12.—Body emits a foul odor; surface covered with petechiæ; intellect dull; tongue soft and cracked; blood oozing from cracks in tongue; tip and edges of tongue red; center coated with yellow fur. Conjunctiva yellow and injected. Capillaries of surface congested, giving a dusky, mottled appearance. Urine scant and contains albumen, bile and casts of tubuli uriniferi, and few cells. Nausea and vomiting; vomited matter streaked with blood specks. Treatment: Ice-water injections; ice-bag over epigastrium. Brandy diluted with water charged with carbonic acid. Iced milk and beef tea.

October 13.—Patient dull and stupid; action of kidneys feeble. Condition of intellect due in part at least to failure of function of kidneys and retention of excrementitious matter. Ice-water injections have increased to a small extent the excretion of urine, but the amount is still far less than normal. Urine contains albumen, bile and casts of tubuli uriniferi. Patient has thrown up black vomit in considerable quantities. Black vomit acid and contains urea. Repeat cold water injections and ice to epigastrium.

October 14.—Patient dull and lethargic in the exercise of the intellect. Talks incoherently; very restless; attempts to get out of bed and speaks of going home. Has passed a small quantity of urine, albuminous and loaded with casts (granular and yellow), and also contains bile. Great capillary congestion of surface; jaundice; spits up black vomit against the wall and over the bed like tobacco juice; the black vomit appears to come up without any effort. Can retain nothing upon his stomach.

Died October 15, 6 A.M.

During the night of October 14 the patient discharged black vomit from his mouth, over his bed and against the side of the wall. The black vomit appears to come up apparently without an effort. Surface of the body of an intense yellow, with great capillary congestion. Only a small quantity of urine was collected from the 12th up to the time of death, although the nurse states that the patient has "passed his water in the bed," after the administration of the ice-water injections. The application of bags of ice to the epigastrium did not arrest the nausea and vomiting. The sluggishness of intellect and delirium increased progressively from October 12.

Post-mortem examination, three hours after death: Body of a golden color; lower portion of a purplish, mottled appearance from capillary congestion. Dark brown blood, black vomit, issues from the corners of the mouth. Blood issues from the left ear. Thorax: Lower portions of lungs congested; otherwise normal. Heart: Muscular structures of heart present a yellow appearance, as in fatty degeneration of this organ: Under a magnifying power of 450 diameters, the transverse striæ of the muscular fibers were found to be indistinct, and the fibrillæ were filled with oil globules, and granular albuminoid or fibroid matter; the oil globules were also deposited around the fibrillæ. The albuminous substance of the sarcous elements of the fibers were evidently in an altered and degenerated state, being resolved into granular matter and fat. Heart still warm, and its fibers filled with dark fluid blood, which coagulated and changed to the arterial hue when removed and placed in glass vessels. Under the microscope, with a magnifying power of 450 diameters, the vast majority of the colored blood corpuscles presented a normal appearance, while a few presented a crenated and stellate appearance. The blood corpuscles adhered together, forming rolls, as in the blood of inflammation. The blood contained oil globules. The dark pigment particles, as well as the colored corpuscles charged with pigment particles, characteristic of the blood in true malarial paroxysmal fever were entirely absent. After careful microscopic examination, I discovered no living organisms, neither bacteria, fungi, algæ, or animalculæ of any description in the blood. Reaction of the blood very slightly acid. Fibers diminished in amount. Clot voluminous and soft. Upon standing, a small amount of golden-colored serum, containing also some colored corpuscles, was pressed out of the coagulum; the fibrin of the clots, however, manifested very feeble contractile powers. Pericardium greatly congested, the smallest vessels being distinctly seen, filled with red blood. This congestion of the pericardium appears to be characteristic of yellow fever. Abdominal cavity: Liver presented a yellowish mottled appearance. The mottled appearance of the liver was due to the congestion of the hepatic capillaries within the central portion of each lobule.

The periphery of each lobule, in the area occupied by the portal system of capillaries, presented the deep yellow color characteristic of the liver in yellow fever. Upon careful inspection of the surface, and of sections of this organ, many portions were found to be without any marked capillary congestion of the hepatic capillaries, and presented a uniform yellow color. Upon a superficial examination of this organ, as it lay in its natural position in the abdominal cavity, the appearance resembled that of the liver of health, the color approaching Spanish brown; when, however, it was carefully examined, within and without, the characteristic change induced by the disease was evident; and when sections were subjected to the gentle action of water, and the excess of blood pressed out of the capillaries the color was uniformly yellow, as in the livers of those who had died at a more advanced stage of the disease.

The depth of color of the liver in this case was due to two causes: 1, to the great congestion of the hepatic (central) capillaries; and 2, to the presence of dark granular masses of hematin, scattered chiefly through the meshes of the portal system of capillaries, and which had evidently been deposited during the preceding attacks of malarial paroxysmal fever.

It is probable that such cases as the one now under consideration have led superficial observers to assert that the liver presents no characteristic lesion in yellow fever. Under the microscope, the liver was observed to be loaded with oil globules. Fatty matter in the form of oil globules of various sizes, was accumulated within and between the cells. The structures of the liver were firm, and the blood also changed to the arterial hue upon exposure to the atmosphere. While, therefore, the microscopic examination revealed the effects of preceding malarial disease, at the same time, it also clearly illustrated the pathologic changes induced by the yellow fever poison. The muscles of the walls of the abdomen and of the chest, when exposed to the atmosphere assumed a brilliant scarlet color, wholly unlike the dark color of the muscles in malarial fever.

Gall bladder entirely empty; contained no bile. Kidneys: Congested and of a yellow color, resembling the color of the heart and liver. The yellow color of the kidneys was especially evident when sections were washed under a gentle stream of water. Sections of the kidneys, with Valentine's knife, examined under magnifying powers of various degrees, revealed the presence of oil globules, granular albuminoid or fibroid matter, and detached excretory cells within the Malpighian corpuscles and tubuli uriniferi. The blood vessels of both the cortical and medullary portions were congested with blood. Both the cortical and medullary portions presented a yellow granular appearance. Many of the uriniferous tubes presented a yellow opaque appearance, being completely filled with yellow granular matter, oil globules of various sizes and detached epithelium. This yellow opaque matter resembled in all respects that composing the casts of the tubuli uriniferi found in such abundance in severe cases of this disease. In some tubules the epithelial cells could not be discerned, either because they had been detached and removed, or else because they were obscured by the oil and granular matter with which the structures were infiltrated. In portions of the kidneys the oil globules were observed around as well as within the tubuli uriniferi.

Bladder: The urinary bladder contained about eight fluid ounces of golden or orange-colored urine.

Reaction of urine, acid; specific gravity 1012. Albumen present in considerable amount. Under the microscope, detached cells from the tubuli uriniferi and yellow granular casts were observed in considerable numbers; but as usual there were no crystalline forms of urinary deposits. No living animal or vegetable forms were observed in the urine. Urine contained no grape sugar.

Alimentary canal: Stomach and intestines somewhat congested upon the exterior. Mucous membrane of stomach moderately congested in some portions, and in others patches were observed of a deep red color; it was also denuded of epithelium in some parts.

Black vomit: The stomach contained about eight fluid ounces of thick, grumous, dark, purplish black, bloody looking black vomit. Under the microscope the black vomit was found to contain some oil globules of the milk administered to the patient, with numerous black masses of hematin, altered blood corpuscles, epithelial cells and patches, and fibroid and granular casts of the mucous membrane of the stomach and broken capillaries.

The black vomit, although taken from the stomach only three hours after death, and while the body of the subject was warm, emitted a foul, putrid odor, as of decomposed blood. Upon careful microscopic examination I discovered no animalculæ or vegetable plants (fungi or algæ) in the black vomit. Reaction of black vomit slightly acid.

The spleen was enlarged and somewhat softer than in health, but much firmer than in recent cases of malarial paroxysmal fever.

CASES OF YELLOW FEVER, OBSERVED 1873, BY DR. TAYLOR, M.D., OF NEW ORLEANS.

Joseph Fitzpatrick. aged 6 years; taken sick September 21. Had spent more than one summer in the city. Recovered. September 21, pulse, morning, 156, evening, 148; Sept. 22, morning, 128, evening, 120; Sept. 23, morning, 108, evening, 104.

Date.	Pulse.		Resp.		Temp. Cent.	
1873	M.	E.	M.	E.	M.	E.
Aug. 17	112	124	....	....	....	40.2°
" 18	118	104	....	....	39.2°	38.6°
" 19	104	104	....	....	36.6°	39 °
" 20	106	116	....	....	39.2°	40.2°
" 21	108	100	....	....	39 °	39.8°
" 22	108	120	....	....	39.8°	40.2°
" 23	108	128	....	....	39.4°	40.4°
" 24	120	136	....	....	....	....
" 25	132	128	....	....	....	....
" 26	124	128	....	....	....	....
" 27	120	116	....	....	....	....
" 28	110	112	....	....	....	....
" 29	96	88	....	....	....	....
" 30	84	76	....	....	....	....
" 31	66	56	....	....	....	....
Sept. 1	48	40	....	....	....	....

HISTORY OF CASE. REMARKS.

Jas. Crawford, aged 8 years, taken ill Aug. 16. Saw him for first time on evening of Aug. 16. Crawford children had arrived from Ireland only three months previously. In this case, as soon as remission and exacerbation began to be distinctly marked (say from about the seventh day), I began using quinia in gr. xx doses in 24 hours. The remission and exacerbation evidently were not affected by the drug for several days, and then variations continued for some time after Aug. 23, when, owing to an accident, I was compelled to suspend the use of the thermometer. I gave quinin from the morning of the twelfth day. The condition of the stomach remained good nearly all the time. I gave beef tea and milk several times each day. In this, although the symptoms and appearance of the patient pointed strongly to yellow fever, and although the urine contained albumen, still the indications afforded by the pulse and thermometer caused hesitation. Quinia, however, although freely used, did not bring about any solution to my doubts, and I finally suspended its use. The fever really seemed to decrease after the twelfth day, and the minimum of pulsations (40) was reached only at the close of the eighteenth day. For several days during the height of the disease I was considerably annoyed by a free discharge of blood from the gums, which was at last checked by the internal use of the perchlorid of iron. Convalescence was tedious, owing to the great debility of the child, who suffered from several carbuncles attended by sloughing.

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Sept. 20	120	112	....	....	....	....
" 21	108	92	....	....	....	104.6°
Night	. .	92	....	....	....	103.6°
" 22	96	112	....	....	....	....
Night	....	120	....	....	....	....

REMARKS.

Father Schneider; taken ill September 20, about 2 P.M.; died on September 23, at 9 A.M. Black vomit. Had spent two years in St. Thomas; came to New Orleans March, 1870. Immediately after his death the bed and bedding and his clothes were burnt; the room and house disinfected a few hours later by the board of health.

Date.	Pulse.		Resp.		Temp. Cent.		REMARKS.
1870	M.	E.	M.	E.	M.	E.	
Sept. 8	104	96	20	24	40°	40.8°	Luce Maxime; taken with fever September 6, 1870. Paid first visit on September 7, 1870. No trace of albumen in urine which was freely excreted during the whole attack; recovered.
" 9	88	88	20	20	40.6°	41°	
" 10	80	76	32	24	40.2°	40.2°	
" 11	64	60	24	20	39.4°	39.6°	
" 12	60	64	20	20	38.6°	38.8°	
" 13	60	56	20	20	38°	37.8°	
" 14	52	52	20	20	37.6°	37.6°	
" 15	52	48	20	20	37.6°	37.6°	
" 16	41	56	20	20	37.8°	38°	
" 17	66	64	20	24	38°	38°	
" 18	50	64	20	20	37.8°	37.6°	
" 19	56	48	20	24	37.8°	38°	
" 20	64	68	24	20	38°	38.2°	

Date.	Pulse.		Resp.		Temp. Cent.		REMARKS.
1870	M.	E.	M.	E.	M.	E.	
Sep. 28	116	116	. .	. .	C.	C.	M. T. Bugman; age 22 years; recently arrived from Kentucky; laborer, taken ill September 27, 1870; first visit made September 28. Jaundice and suspicious character of vomit noted on 29th; great quantity of black vomit on September 30 and October 1. Died in convulsions at 3 P.M. October 1.
" 29	96	100.	. .	. .	40.4°	41.2°	
" 30	92	92	. .	. .	40.4°	41.2°	
Oct. 1	100	. .	. .	. .	38.8°	40°	

Date.	Pulse.		Resp.		Temp. Cent.		REMARKS.
1870	M.	E.	M.	E.	M.	E.	
Sep. 29	72	72	. .	. .	38.6°	38.6°	No diminution in quantity of urine; no albumen perceptible; recovered
" 30	68	68	. .	. .	38.8°	40°	
" 31	68	68	. .	. .	38.2°	38.4°	
Oct. 1	72	68	. .	. .	38.4°	38.6°	
" 2	60	64	. .	. .	37.6°	37.8°	
" 3	54	54	. .	. .	37.4°	38°	
" 4	50	54	. .	. .	37.6°	37.8°	
" 5	50	54	. .		37°	37.4°	
" 6	50	. .	. .	. .	37.4°	37.4°	
" 7	60	. .	. .	. .	. . .	. . .	

CHAPTER IV.

POST-MORTEM TEMPERATURE IN YELLOW FEVER.

The medical profession is indebted to Dr. Bennet Dowler, of New Orleans, for the first, and up to the present time the most extensive, observations illustrating the temperature of various portions of the body after death from yellow fever.

The results of the researches of Dr. Dowler were originally transmitted to the *Western Journal of Medicine and Surgery*. during the winter of 1843-44, but were returned to the author unpublished. The original data were published sixteen years later by Dr. Dowler in the *New Orleans Médical and Surgical Journal*, March, 1860, Vol. XVII, No. 2, pp. 199–220, and from this paper I have consolidated the following tables, which will illustrate the state of our knowledge with reference to the post-mortem temperature in yellow fever:

Temperature of the primary period of those who died of yellow fever.

Hours Sick.	Hand.	Axilla.	Observations.
12	102°	105°	
12	103°	107°	
12½	105°	105°	Died in seven days.
24	104°	107°	Died in four days.
28	107°	107°	
15	. . . .	102°	
37	97°	102°	
26	104°	109°	
29	107°	109°	
34	103°	104°	
42	97°	102°	
24	99°	100°	Died in two days.
24.6	102.5	104.9	

Temperature of middle period of those who died of yellow fever.

Hours Sick.	Hand.	Axilla.	Observations.
1	99.5°	104°	
5	101°	104°	
5	97°	102°	
3½	100°	105°	
3½	100°	105°	Died in three days.
2	100°	102°	Died in four days.
4	104°	107°	Died in three days.
3	100°	104°	Died in three days.
4	102°	105°	
5	91°	102°	
6	100°	105°	
6	102°	105°	A relapse.
7	98°	100°	Died in four days.
8	93°	99°	Died in three days.
5	99°	104°	
8	102°	102°	
12	106½°	105°	
1	99°	100°	
6	100°	104°	Died in two days.
7	91°	102°	Died in three days.
7	101°	104°	
8	102°	102°	
12	105°	106½°	
14	101½°	103°	Died in three days.
6	99°	. . . .	Died in three days.
6.4	99.4	103.39	

Temperature in the primary period of yellow fever in persons who recovered.

Age.	Hours Sick.	Hand.	Axilla.	Observations.
29	3½	102°	106°	V. s. ad del.; cured in six days.
28	20	102	105	V. s. ad del.; well in six days.
24	8	101	103	V. s. ad del.; well in seven days.
30	9	100	106½	Cured in thirty-eight days.
. . . .	5	102	104	Intermittent type; well in eight days.
30	12	105	105	V. s. ad del.; cured in six days.
35	13	102	104	Cured in seven days.
24	14	102	104	Cured in ten days.
27	13½	102	104	Cured in twenty days.
21	16	107	107	Cured in seven days.
28	19	102	104	Cured in three days.
29	22	102	104	V. s. ad del.; well in four days.
25	28	102	106	Cured in nine days.
28	26½	102	104	V. s. ad del.; cured in fourteen days.
41	48	99	102	
30	48	98	104	V. s. ad del.; cured in one day.
22	48	104	106	
21	48	. . . .	102	Ninth day convalescent.
28	48	97	103	Ninth day cured.
26	48	105	. . . . .	Cured in ten days.
29	48	97	102	Cured in six days.
23	60	. . . .	103	Cured in seven days.
34	24	99	102	
31	14	95	102	Cured in twenty days.
25.5	26.8	101.2°	104.1°	

Temperature in the fatal stage of yellow fever, with some account of the respiratory action.

Hours before Death.	Hand.	Axilla.	Respiration.
10	101°	102°	Respiration quick and uneasy.
24	95	104	Respiration quick.
8	. . . .	101	Coma; seven respirations per minute.
1	91	100	Respiration tired, heaving.
1½	94	100	Respiration noisy, quick, laborious.
6	89	100	Respiration noisy, slow.
2¼	70	95	
9	94	100	Respiration imperfect, quick, irregular.
21¼	95	100	Respiration noisy.
21½	104	106	Respiration irregular.
11½	81	90	
11	88	100	Respiration quick, wheezing; coma.
10	84	95	Respiration easy.
7½	86	97	Respiration laborious, irregular.
12	95	102	Stertor; coma.
11	89	95	Respiration quick and irregular.
12	. . . .	99	Respiration noisy, quick and laborious.
6	83	. . . .	Respiration imperfect, small, irregular, quiet.
7	81	90	Respiration extremely laborious, gasping.
26½	104	105	Coma; stertor.
24	. . . .	100	Respiration hurried; coma.
21½	97	104	Coma; stertor.
23	89	98	Respiration quick.
1	84	95	Respiration slow; loud heaving.
67½	101½	103	Respiration imperfect.
5¼	100	101	Respiration quick and irregular.
12	102	102	Respiration quick.
40	. . . .	97	Respiration quick.
24	89	100	Respiration loud, laborious, rattling; coma.
1	. . . .	102	Respiration loud, puffing.
2	. . . .	100	Respiration loud, quick; coma.
48	88½	97½	Respiration quiet, puerile.
2½	89	99	Respiration stertorous; coma.
24	93	97	Respiration hurried, unequal.
28	102	104	Respiration easy.
72	88	101	Respiration quick, very easy.
5	. . . .	95	Respiration rapid, small.
9	. . . .	98	Respiration hurried, suffocative.
4½	89	97	Respiration easy.
24	91	101	Respiration imperfect; nineteen per minute.
24	88½	102	Respiration wheezing, puffing; rattling cough.
2	93	97	Respiration irregular, laborious, quick.
24	66	95	Respiration quick, puerile.
12	93	101	Respiration quick, puerile, loud.
12	. . . .	100	Respiration hurried.
30	104	106½	Respiration hurried.
4	102	104	
6	. . . .	96	Respiration slow, imperfect.
12	100	101	Respiration fifty-seven; coma.
24	100	103	Respiration stertorous; comatose.
5	89	100	Respiration noisy, slow.
16.6	92.2	99.5	

Temperature of the middle period of yellow fever of those who recovered.

Age.	Days Sick.	Hand.	Axilla.	Observations.
30	6	106 °	108.2°	Third day convalescent.
27	2	102	105	
30	1½	99	105	
. . . . .	2½	102	102	
29	2	100	102	Cured in three days.
34	4	102	104	Cured in ten days.
. . . . .	5	. . . . .	102	
21	2	97	103	Cured in nine days.
26	6	97½	100	Cured in five days.
24	14	100	104	
. . . . .	16	106	106	
24	3	97	102	Cured in thirty days.
29	5	102	109	Cured in twelve days.
38	4	100	102	Cured in two days.
23	4	100	104	Cured in fifteen days.
19	16	104	104	Cured in five days.
38	4	104	104	Cured in nine days.
38	12	95	102	Cured in fifty days.
27	4	99	104	Cured in four days.
22	9	103	104	Cured in twenty-eight days.
34	5	99	100	Cured in six days.
20	4	97	100	Cured in five days.
32	7	100	103	Cured in ten days.
24	5	99	100	Cured in six days.
32	6	100	102	Cured in six days.
22	3	100	103	
22	4	100	104	Cured in four days.
20	4	100	107	Cured in four days.
25	6	107	107	Cured in eight days.
28	6	102	102	Cured in seven days.
30	8	101	102	Cured in five days.
18	7	102	104	
21	9	101	102	Cured in sixteen days.
23	. . . . .	97	101	Cured in twelve days.
27	4	104	108	Cured in seven days.
35	12	91	102	
22	7	93	100	Cured in six days.
18	11	102	102	Cured in four days.
24	9	94	97	Cured in ten days.
41	2	99	102	
26.9	5.9	100.4	103.1	

Temperature of the period of convalescence in yellow fever.

Days Sick.	Hand.	Axilla.	Days Sick.	Hand.	Axilla.
2	100 °	99 °	5	. . . .	95
5	96	100	16	97	98
3	100	102	10	100	100
4	93	99	18	95	98
5	91	97	8	. . . .	102
4½	99	98	7	97½	101
2½	86	97½	8	99	101
1½	97	100	1	98½	102
9	. . . .	97	. . . .	100	102
7	97½	83	2	98	100
9	94	100	5	95	99
6	95	99			
8	96	99	6.5	96 °	98.46°
9	93	95			

Post-mortem series, showing the post-mortem heat of regions. Death caused by yellow fever.

Time dead when observations began.		Time observations lasted.		Air.	Axilla.	Thigh.	Rectum.	Perineum.	Pelvis and Abdomen.	Liver.	Epigastrium.	Chest.	Heart.	Brain.
H	M	H	M											
	10	4	8	90½	109°	113°	...	...		112°	111°	...	109°	...
	5	2	.	89	107	107	105°	...	106°	106	105	106°	104	...
	30	.	33	88	104	...	112	102°	...	...	103	...	...	102°
	15	1	5	79	102	102	104	...	...	...	101	100	100	...
	5	.	31	70	106	102	...	...	...	...	106	...	...	101
	10	1	.	71	96	98	...	...	...	...	90	97½	...	93
	50	1	.	82	103	102	...	...	...	.	104	...	...	100
3	. .	2	.	80	101	100	100	...	...	102	101	...	...	95
1	. .	1	9	82	103	104	...	...	...	108	104	97	...	99
	10	1	51	80	106	108	...	...	...	...	106	...	...	101
	10	2	20	84	106	107	...	...	...	...	109	...	...	...
3	. .	.	51	85	106	105	...	...	...	...	109	107	...	...
2	. .	2	.	80	102	103	...	...	...	...	104	103	...	...
	20	1	40	90	106	106½	...	104	...	...	106	105	...	...
	30	.	45	86	103	100	...	...	...	...	105	...	...	...
3	. .	.	10	82	106	...	105	...	...	...	...	...	...	...
1	. .	2	.	84	106	106	...	...	...	...	106	106	...	...
	33	.	33	90	108	...	...	106	...	...	...	...	...	...
2	. .	1	41	91	. .	103	102	...	...	...	104	101	...	...
	5	1	8	86	104	108	104	...	...	...	106	103	...	...
	15	1	45	88	104	106	...	...	...	...	109	...	106	...
	15	2	.	89	104	106	109	...	...	...	108	104	103	...
1	30	.	20	82	104	...	104	...	...	...	...	...	...	...
	15	1	45	82	102	104	101	...	102	105	104	102	99	...
	20	3	20	83	108	109	...	104	107	104	109	104	...	...
2	. .	1	30	80	105	...	104	...	...	...	105	...	...	...
2	30	3	50	86	109	...	107	109	107	107	105	102	102	...
	30	1	4	83	107	108	...	...	106	...	109	102	102	...
	30	1	40	87	105	...	104	104	107	...	...	...	...	...
	1	4	.	90	103	102	...	102	104	104	104	105	105	...
	30	4	.	90	106½	...	111	109	...	...	...	...	...	...
1	. .	3	.	89	103	...	102	104	...	...	...	...	...	...
	15	4	45	86	107	109	...	104	106½	109	108	106½	106½	...
	15	1	.	82	102	100	...	...	...	...	102	...	...	...
	16	2	.	84	104	103	107	...	...	...	110	...	...	...
	10	.	.	85	107	107	...	...	...	...	107	104	...	...
	10	.	26	85	103	104	...	...	...	...	...	102	...	...
	5	.	17	75	107	107	...	...	...	...	...	...	...	...
	30	.	20	87	100	...	...	...	100	...	...	102	...	...
2	. .	.	35	84	100	...	98	...	...	...	...	...	...	...
	25	.	55	85	103	...	102	101	...	...	...	...	...	...
	5	.	12	90	106	...	106	...	...	...	...	...	...	...
	10	.	36	85	103	104	...	...	...	...	102	...	102	...
M	58½	8	32	84.4	104.44	104.44	104.05	104.95	105.05	106.33	105.48	102.95	103.5	98.71

Post-mortem series, showing the decline of post-mortem heat, the antagonism of the surrounding media or incipient regional refrigeration, the forerunner of putrefaction.

Time dead when observations began.		Duration of Experiment.		Air.	Epigastrium.	Lungs.	Heart.	Liver.	Rectum and Pelvis.	Thigh.	Axilla.	Brain.
H.	Min.	H.	Min.									
3	20	. .	40	81 °	104 °	104 °	. . .	. . .	104 °	. . .	.	. . .
4	50	1	10	86	105	. . .	102 °	107 °	102		. . .	. . .
4	. . .	. .	15	82	100	93	. . .	. . .	. . .	99 °	. . .	. . .
10	. . .	. .	9	90	93	. . .	. . .	. . .	. . .	92	. . .	. . .
2	. . .	. .	10	84	106	. . .	. . .	. . .	. . .	103	. . .	. . .
3	. . .	. .	15	85	. . .	104	. . .	. . .	. . .	100	. . .	. . .
5	. . .	. .	28	66	93	. . .	. . .	. . .	. . .	88	88¼	86 °
4	30	. .	30	70	100	97	. . .	. . .	. . .	99	. . .	93
12	. . .	. .	55	79	92	91	91	. . .	91	89	. . .	84
11	. . .	.	25	80	96	. . .	93	. . .	. . .	93	. . .	85
6	. . .	. .	8	91	102	. . .	. . .	. . .	. . .	100	. . .	. . .
3	. . .	. .	20	86	104	. . .	. . .	. . .	. . .	100	100	. .
13	. . .	. .	10	80	95	. .	. . .	. . .	. . .	93	. . .	. . .
6	. . .	. .	20	80	103	. . .	. . .	. . .	. . .	100	100	. . .
15	. . .	. .	30	86	92	91	. . .	. . .	. . .	87	. . .	. . .
10	. . .	. .	20	80	91	. . .	. . .	. . .	. . .	86	. . .	. . .
3	. . .	1	. . .	61	84	80	87	86	. . .	84	. . .	. . .
6			27	86.29	97.5	94.28	93.25	96.5	99	94.26		

Post-mortem series, showing the coincidence of temperature between the dead body and the atmosphere at the period of incipient putrefaction.

Hours Dead.	Temp. of Room.	Epigast.	Lungs.	Brain.	Axilla.	Thigh.
16	87 °	89 °	89 °	. . . . .	. . . . . .	89 °
23	90	92	92	. . . . .	. . . . . .	88
14	80	83	84	78 °	. . . . . .	81
19	86	89	89	. . . . .	. . . . . .	86
13	88	95	. . . . .	. . . . .	. . . . . .	93
27	86	86	. . . . .	. . . . .	. . . . . .	82
20	91	91	. . . . .	. . . . .	. . . . . .	91
16	71	. . . . . .	. . . . .	74	. . . . . .	88
10	90	93	. . . . .	. . . . .	. . . . . .	92
15	90	93	. . . . .	. . . . .	. . . . . .	88
15	86	92	91	. . . . .	. . . . . .	87
19	88	91	. . . . .	. . . . .	. . . . . .	88
18	88	90	. . . . .	. . . . .	. . . . . .	88
22	90	90	. . . . .	. . . . .	. . . . . .	. . . . .
12	70	. . . . . .	. . . . .	. . . . .	86 °	. . . . .
6	91	. . . . . .	. . . . .	. . . . .	91	. . . . .
24	54½	65¾	. . . . .	. . . . .	. . . . . .	56¾
17¾	83	. . . . . .	. . . . .	83	. . . . . .	89
. . . . .	94	82	. . . . .	81½	82½	79
17.04	84.3°	88.1°	89°	79.12	86.5	85.35°

Post-mortem series, showing the comparative temperature taken at variable periods after death, in several regions of five different bodies, amounting to forty maxima.

Maxima of eight regions compared in five cadavers.

Thigh.	Epigas.	Axilla.	Chest.	Heart.	Brain.	Rectum.	Liver.
113°	111°	109°	107°	109°	102°	111°	112°
109	110	109	106½	106	101	109	109
109	109	108	106	105	101	107	108
108	109	108	106	104	100	107	107
108	109	107	105	104	99	109	106
M 109.4	109.6°	108.2°	106.1°	105.6°	100.6°	108°	108.4°

It will be observed that Dr. Dowler records three cases in which in the primary stage of yellow fever, the temperature of the axilla reached 106 degrees; one 106.5 degrees, and one 107 degrees; in the middle period, one case 106 degrees, two cases 107 degrees, one case 108 degrees, one case 108.5 degrees, and one case 109 degrees; and notwithstanding the high temperatures the patients recovered. The patient whose temperature reached 108 was cured in seven days; the one with a temperature of 108.5, was convalescent on the third day; and the third with the axillary temperature at 109 degrees, was cured in twelve days. The highest temperature observed by Dr. Dowler in fatal cases of yellow fever was 110.5 degrees axilla. On the other hand, by boldly exploring the regions of the body, a few moments after death, Dr. Dowler has shown that the temperature of the deep-seated muscles of the thigh may attain a temperature of 113 degrees F., the epigastrium and rectum 111, and the liver 112 degrees.

There is no proof furnished by the observations of Dr. Dowler that there was an actual increase of heat in the internal organs after death above that which it may have attained during the active stage. All the facts recorded in his tables appear to sustain the view that the high temperatures existed at the same time during life in the internal organs. Thus in those cases in which the temperature of the surface of the body in the region of the axilla reached 110.5

during life, it is probable that the temperature of the internal organs was at least from 2 to 4 degrees higher; and it is worthy of note that in not one of his observations on the post-mortem heat of regions, did the temperature of the axilla exceed 110.5, the highest point reached during life. It is still further worthy of note, that the case in which the thigh gave 113 degrees, the liver 112 degrees, epigastrium 111 degrees, and the heart 109 degrees, the temperature of the axilla was 109 degrees.

My own investigations upon the post-mortem heat in yellow fever sustain the view that there is no actual increment of heat above the highest maximum of the internal organs during life. With the sudden cessation of respiration and of the consequent transpiration from the surface of the lungs, and the sudden suppression of the perspiration of the skin and the consequent diminution of evaporation from the surface, there must necessarily be a change in the distribution of the heat, and as it is diffused by conduction from the center to the circumference, an increment of heat on the dead surface. If, however, this view be incorrect and if there is in certain cases an actual increment of the total amount of heat in the body, after death by yellow fever, such increment is probably due to the following causes: 1, cessation of respiration and evaporation from the surface of the lungs; 2, cessation of perspiration and diminution of evaporation from the surface; 3, the continuance of chemic changes chiefly in the blood and internal organs, and the consumption of the oxygen of the blood for short periods after death, the transformation of certain modes of force, as the nervous and muscular into heat.

To demonstrate conclusively whether there is an actual increment of heat in the body after death from yellow fever, comparative experiments should be instituted by immersing those suffering with yellow fever in definite amounts of cold water, (the quantity being regulated in accordance with the weight of the patient), and noting carefully the temperature assumed by the surrounding water at definite periods. Similar experiments, corresponded exactly in the quantity and temperature of the water employed and in the periods of time. The main element of disturbance to be calculated and as far as possible, eliminated from the first series of experiments would be the effect of pulmonary acts or respiration. In the absence of such exact experiments, the subject of post-mortem heat in yellow fever and other diseases must necessarily be involved in obscurity.

Dr. Dowler, however, inferred from his experiments that there was an actual increment of heat in the body after death from yellow fever, and the views of this distinguished observer and physiologist are worthy of the most careful consideration. Thus in commenting on the results of his investigations he says:

"The average of five *maxima* of the epigastrium are nearly the same; but if we select ten *maxima* from each of these regions, the thigh will be found the hotter of the two by 0.3 degrees. This is the more remarkable, as the observations were made, owing to the emergencies incidental to an epidemic, at irregular, sometimes at lengthened periods after death, when the thigh more especially, from its comparative smallness, and greater exposure, had parted with more or less of its morbid caloric, by contact and radiation.

"In one series of cases of yellow fever, taken without selection, the following results were obtained: Fifteen patients who recovered, when temperatures were taken at a period which averaged fifteen and one-third hours, after the invasion, and afforded a mean temperature of the hand of 101.8, and for the arm-pits 104.84 degrees; nine persons who died, gave at an average of twenty-two and one-third hours after the invasion—for the hand 103.62 degrees; for the arm-pits 105.44 degrees.

"The maxima of these classes coincided, but not the minima. In those who died the average was higher; in the hand, the maximum reached by both was 107 degrees; the minimum in the hand among those who recovered, was 95 degrees, and of those who died 99 degrees; the maximum of the former in the axilla, 107 degrees; of the latter 109 degrees; the minimum of the former in the same region, 102 degrees, and of the latter 100 degrees, the latter being *in articulo mortis.*

"The extraordinary fact that in some bodies recently dead, the temperature, at various periods thereafter, rises higher than it had risen during any stages of the maladies which preceded—much higher than in the latter stages—has been fully confirmed, as well as another, still more extraordinary; namely, that at uncertain periods, usually, perhaps less than an hour, though occasionally later, the center and periphery will attain to, and remain at a stationary, perhaps a high temperature, which, for a considerable time will neither rise nor fall. But, still further than this, either the center or circumference, or both together, having reached a certain temperature, will sometimes fall and rise several degrees, repeating these movements several times. These internal and external fluctuations may or may not coincide in time, degree, and duration. Assuming that these thermal currents originate in different regions, independently of each other, and pursue different, perhaps curved routes, occasionally uniting at certain points or foci, it might be expected that at these points of contact or convergence the maximum heat would be found. These foci are not found in a marked degree, even in the center of the brain, far less in the inferior extremity below the knee, when the calorifacient power is most feeble or null in cadavers.

"How hot soever the patient may have been during the progress of fever, the heat generally recedes before and at death; and this recession will, upon averaging a great number of cases approximate natural standards, at least in the arm-pits and some other accessible regions.

"The development of post-mortem calorification does not appear to be materially accelerated or retarded by the atmospheric temperature, humidity, or dryness. This heat is not the effect nor the accompaniment of, but antagonistic to putrefaction. When calorification ceases, physical refrigeration begins; and when the latter is reached, putrefaction is rapidly developed, if the weather be sufficiently warm. The laws of post-mortem calorification are numerous and complex. Its increment, decrement, degree, duration, and repeated ebbings and flowings, differ in different bodies, so much that neither physics nor physiology has as yet furnished any satisfactory standard explanation but a parallelism. Periodic diseases, in which paroxysms of cold and heat alternate, seem to indicate analogy, if not identity, as do the great, but little known fluctuations of the normal temperature during morning, noon, evening and night, among persons in health." (*New Orleans Medical and Surgical Journal*, March, 1860, Vol. XVII, pp. 210–212.)

In the May number of the same journal, in which Dr. Dowler continues the subject, he remarks:

"Both physiology and pathology furnish abundant evidence that animal heat is due neither to pulmonary combustion, nor in any way attributable to the energy of respiration. For example: In solar asphyxia (sunstroke of the first degree), which is almost always quickly fatal, respiration is very imperfect, being restricted chiefly to the bronchii and trachea, and is attended with loud mucous rattlings (râles); yet this is the hottest of all diseases. As the suffocation, I might say strangulation, increases, so does the heat, which in a few minutes reaches in some cases, 112 degrees F. in the axillæ and other regions. The post-mortem heat is equally remarkable, and may be of very long duration. The lesion usually found being either extravasation into the pulmonary tissues or congestion, which illustrates the previous history of pulmonary embarrassment.

"In sudden apoplexy (*apoplexie foudroyante*), as well as in most cases of apoplexy, coma occurs, and as the coma increases, the heat increases and is very persistent after death. Generally, in cases of the comatose form of death from acute diseases, particularly fevers, the close of life is attended with comparatively high temperature, notwithstanding the embarrassment and infrequency of the respiratory act." . . . . "Whether the pulmonary congestion be the primary or secondary condition of insolation, I will not say; but I must remark, that of all morbid appearances of a congestive character, this is the least equivocal, so far as I have examined. . . . . Although physiology teaches us that man is endowed with the power of maintaining the same heat of his body in all climates and all situations, with few exceptions, still it is possible, under peculiar circumstances, that the body may become actually heated beyond the normal standard. A chemico-vital refrigeration by means of perspiration or evaporation, is constantly going on. 'The fire kings,' themselves, when in a heat of 500 or 600 degrees, would roast and turn to cinders were it not for this refrigerating process, in conjunction with a vital energy which for a time neutralizes the accumulating power of caloric. The solar heat in sunstroke probably accumulates upon the surface (and in the lungs), of the body faster than nature can refrigerate through the lungs and the skin by evaporation; the vital energy being exhausted by the contest as well as by the excessive labor (in most cases), is unable longer to neutralize the excess of temperature; vital chemistry is unequal to the task of preventing the conduction of heat into the body, and death is the consequence." . . . . "If we suppose the central, the great vital organs to be as hot during life as they are found to be after death, the only wonder is that vitality should maintain its seat for a week or more, under the positive changes that ought, by every law of calorics, to take place in the molecular arrangement of the tissues. Let us suppose the brain in life to become as hot as the thigh is found to be after death, that is 14 or 15 degrees F. in health; the cerebral mass would expand faster than its cranial walls; the fluids would dilate and perhaps transude; compression would be the consequence, attended with convulsions, coma, and other effects incompatible with life. Suppose any other organ should become such a focus of morbid caloric only for a moment, would not each vessel from dilatation lose its healthy elasticity and cohesion and thus pave the way to sanguineous congestion? In some diseases, the lesions will afford an average alteration as great as fatal gunshot wounds; as, for example, dysentery, consumption, cancer. But in a fever how much is unexplained! Is not morbid caloric the agent that eludes the knife of the anatomist? To say nothing of its directly poisonous, let us consider its mechanical effects as above mentioned upon the brain. After dilating its delicate vessels, and establishing a sanguineous congestion death follows. The brain, as we have shown, falls sooner than other central parts under the law of refrigeration; the cranium contracts; this tremendous force drives the blood down from the brain towards the warmer and more yielding centers of the trunk; perhaps a real apoplexy, without rupture has disappeared. . . . . So far as morbid heat can be identified as a cause of disease, we deal with a positive, not an imaginary agent, where the ground is not eternally slipping from beneath our feet. Albumen, which abounds in the brain and fluids, coagulates at 160 degrees; hematoxin, the coloring matter of the blood at 149 degrees; and moderate increase of heat vastly augments the solvent powers of the serum over gelatin, so abundant in the body. The phosphorus of the body were it uncombined, would burn in a heat less than 113 degrees.

"Admitting that the whole body be permeated with 10 or 15 degrees of heat, and that it can not render this heat latent, I ask again, is it wonderful that death should ensue? Which atom has not undergone a deleterious modification, or a new arrangement in its chemic, mechanic and vital relations? Delaroche and Berger prove that animals in chambers heated to 120 or 130 degrees F., have their temperature raised 11 or 16 degrees, and die speedily. If, as some maintain, all lesions may be reduced to those of nutrition, caloric is an agent well adapted to play an important and fundamental part, not only diminishing the elementary cohesion of the tissues, but in debilitating all the organs, thereby favoring intertextural depositions, hypertrophies, softenings, hemorrhagic serous effusions, morbid secretions, engorgements, and other alterations, solid, liquid and gaseous." (*New Orleans Medical and Surgical Journal*, May, 1860, Vol. XVII, No. 3, pp. 368–372; *Western Journal of Medicine and Surgery*, April, 1843; *New York Medical Gazette*, Vol. I, p. 209; *New York Journal of Medicine*, 1846.)

The researches of Dr. Bennet Dowler established the important fact that post-mortem heat was not confined to yellow fever, as will be seen from the following cases recorded in his articles on "Animal Heat." (*New Orleans Medical and Surgical Journal*, Vol. XII, 1856, pp. 205, 289, 433, 470, 603, 759):

> "*Case 1.*—Apoplexy, post-mortem heat; E. F. D., aged 46; born in France; for many years residing in New Orleans; stout and fleshy; long a sufferer from strictures and retention of urine; would allow neither the catheter nor the sound to be applied. About a month after my attendance upon him for an attack of acute pneumonia, he was seized in the night with a chill and fever: after rising from his bed in the morning, he fell and instantly expired; in thirty minutes after which, the axilla gave 102 degrees; in ten minutes 105 degrees, continuing stationary twenty-five minutes; in five minutes 104.5 degrees; in five minutes 104 degrees, remaining stationary ten minutes, that is as long as observed. A partial post-mortem examination brought to light a stone in the bladder of enormous size."

In this case it is evident that Dr. Dowler made no observation of the temperature at the moment of death. The rise in the temperature of the axilla may have been due to the equalization of temperature after the arrest of cutaneous and pulmonary transpiration and evaporation. In this case the unknown element upon which the entire theory of the post-mortem increment of heat rests is the temperature of the internal organs and especially of the blood in the large vessels just before death. No such objections can be urged against the observations in the following case which appears to sustain the conclusion that there was a marked increment of heat immediately after death:

Case 1.—Chronic enteritis, with pulmonitis; post-mortem heat. Aug. 18, 1843; room about 80 degrees. M. H., a woman aged 30, sick about sixty days; dead two hours; greatly emaciated; an incision three inches long in the umbilical region, admitting the air freely; temperature in ten minutes, 102 degrees; in fifteen minutes, 104 degrees; in twenty minutes, 106.5 degrees; in thirty minutes, 104 degrees; heart in 20 minutes, 104 degrees; in twenty-five minutes, 104 degrees; in thirty minutes 104 degrees, and slightly declining; right lung infiltrated with pus, gave in ten minutes 102 degrees; thigh 100 degrees.

Case 2.—Typhus; post-mortem heat. Feb. 28, 1848; air of room about 65 degrees. Mrs. ———, aged 26, born in Ireland, last from New York; resident two months; sixteen days sick; dead twenty minutes. Axilla 100 degrees; in five minutes, 101.5 degrees; in ten minutes, 101.5 degrees; vagina in five minutes, 107 degrees; in ten minutes, 107.5 degrees; one hour after death, 107¼ degrees; fifteen minutes later at dark, when the experiment ended, 107¼ degrees.

Case 3.—Typhus; post-mortem heat. July 22, 1845; air at 5 A.M., 76 degrees; at 5 P.M. 94 degrees. A. F., a Swiss, aged 30, last from Havre, ship *Swanton* fifty-one days at sea; died the second day after landing; experiment begun twenty-five minutes after death, and lasted without interruption two and one-half hours, having been stopped by the funeral. At intervals the axilla gave 109 degrees, 110, 110.5, 110¾ degrees: rectum 111.5 degrees; epigastrium 109; left hypogastrium 110; right hypogastrium 110.5; iliac and umbilical 110 degrees.

Case 4.—Typhoid pneumonia with pericarditis-hepatization. May 2, 1844, thirty minutes past noon; air about 80 degrees. P. M., aged 35; sick eight days; hand 90 degrees; bend of the arm 99; axilla 104; popliteal region 99; died five hours after; remained in bed fifteen minutes, and then as long in the dead house, covered with a linen sheet only; thirty-five minutes after death the axilla 104 degrees, and the popliteal 100; in five minutes axilla 105; popliteal 101; in five minutes axilla 105.2; popliteal 101; in 5 minutes axilla 106, popliteal 101.5; bend of arm 101; rectum in five minutes 100; bend of arm 101; in five minutes groin 102; bend of arm 100; in 10 minutes groin 100; hand in ten minutes 100; rectum in five minutes 106; axilla in five minutes 108 degrees nearly.

The following observations were made in the order in which they stand, at intervals of one, two, three or more minutes: Rectum, 108.5; axilla, 101; rectum, 109.5; axilla, 101.5; rectum, 110.5; axilla, 102; rectum, 110.5; axilla, 102. The observations lasted about an hour and a half, and were stopped by the lateness of the evening, at about two hours after death, the air being then about 79 degrees. Brain seventeen hours after death, 83; center of the thigh, 89; air, 83. Putrefaction odor absent at twenty-two hours after death. Here the axilla attained a maxima of 108 degrees, nearly one hour and a half after death, exceeding that of the living state 4 degrees, as taken four and one-half hours before death, exceeding that of the dead subject thirty-five minutes after death by the same number of degrees. A still greater contrast is found in the palm. The rectum rose from 100 degrees in an hour to 110.5 degrees after death, to the maximum of 110.5 degrees at twenty-five minutes later, and was stationary afterward, having gained and retained 10.5 degrees; while the axilla gained 4 degrees, lost 7 degrees and regained 1 degree, when night approached preventing further observations.

Case 5.—Congestive fever; post-mortem heat. Aug. 28, 1848; room 88 degrees. A. F., born in Maine; resident six months; aged 35; sick seven days; dead forty-five minutes; axilla in five minutes, 108 degrees; in five minutes 108.5; in five minutes 109, when the experiment ended.

In the majority of cases of cholera observed by Dr. Dowler, there was after death a progressive refrigeration from the surface toward the center. He records, however, the following case in which there appears to have been a marked increase of heat after death.

Case 6.—Cholera; post-mortem heat. In Aug. 24, 1848; air of the house 78 degrees. H. P., Swiss; dead about thirty minutes; each experiment being consecutive, lasting about five minutes in the following order: Axilla 104.5 degrees; 105.75; tongue 98; rectum 108: 106.5; 106.33⅓; 108; 108.5; 109; epigastrium 108; 110; 110.5; brain through the nostril 103; concave surface of the loin 108; heart 109.5 brain 102.5; abdominal cavity 108; concave surface of loin 109; rectum 103 5. The experiments ended about two hours after death.

Case 7.—Solar asphyxia. coup de soleil, insolation, ictus solis, sunstroke. July 24, 1845; a day of extraordinary heat; air at 6 A.M, 80 degrees; noon 93 degrees. Deaths from sunstroke officially reported ten; unwritten and misnamed by estimation five; total fifteen. G. F., born in Boston; aged 28; some minutes before 6 P.M., fell while at work at the corner of Camp and Julia Streets; thirty minutes after, the experiments began; for twenty minutes, during the agony, the armpits gave 111 degrees; eight minutes after death 112; in fifteen minutes 112, in twenty minutes 112.5; at about 7 P.M. 113. The body was then stripped, laid in a cooling house, exposed to a free circulation of air, and was dressed in a muslin shroud. The axilla had been in the meantime freely exposed, but gave at seven hours and twenty-five minutes 112 degrees; seven hours and thirty-five minutes, 112; at seven hours and forty minutes, 112; at seven hours and fifty-five minutes, 111.5; at eight hours and thirty minutes, 111; at eight hours and forty-five minutes, 110.25; at nine hours, 109.25; at nine hours and fifteen minutes, 109, when the observations ceased.

Dr. Dowler gives the following as his observations of the temperature of this unexampled day: At 6:30 A.M., 84 degrees; at 8, 75; at 7:30, 87; at 8, 88; at 9, 90; at 10 A.M in the sun near a wooden wall, 115 degrees; touching the same, 130; at 11 A.M. sand in the street, 143 degrees; at 2 P.M., sand in the street, 152 degrees; roof of a house, touching, 150 degrees; at 8:30 P.M., 89 degrees. River at 8 A.M., 83.75 degrees; at 3 P.M., 84.5 degrees; in the shade near the sun at 3 P.M,, 102 degrees; in the house 97 degrees; at 4 P.M., 96 degrees.

Case 8.—Solar asphyxia in sunstroke, complicated with serous-meningeal apoplexy. Aug. 30, 1848: 11:45 A.M.; dead house 97 degrees. W. C., Irishman, aged 35; resident one year; ate his breakfast as usual, worked till 9:30 A.M., soon after fell insensible; died 11:20 A.M. Dead twenty-five minutes. The thermometer remained in the axilla fifty-five minutes, without having been changed, as follows: Five minutes, 105 degrees; five minutes, 106.5; five minutes, 108; ten minutes, 108; ten minutes, 108; ten minutes, 108; ten minutes, 108.5; knees, 106; thigh 108; scroto perineal, 108; rectum, seven minutes, 110; fifteen minutes, 110; epigastrium, 109.25; middle of the thigh, 108.75; chest, 107. The small intestines contained from one to two pounds of chyle; thin in and near the duodenum; thick and pasty in the middle and lower third of the jejunum; semi-coagulated but little adhesion; soft clots or flakes of a milky opaline color prevailed in the ileum; in the lower third of the latter, especially near the valve, a point scarcely perceptible here was noticed.

While the above experiments on W. C. were progressing, the veins became greatly distended; a ligature was placed on the arm, a vein was opened, about two ounces of blood filtered out, after which a trickling took place for a considerable time, amounting by estimation to twelve ounces. The circulation was found to be very rapid about the head; the skin of the face and neck were injected, dark, lurid and somewhat mottled; there was no cardiac hyperemia or injection of the dependent parts; the external jugular veins were distended as if ready to burst. The left jugular was opened, as for ordinary blood letting, but no drainage or pressure was used, the head being raised, so that the orifice was nearly on a level with the breast bone. The blood jetted completely without wetting the skin, forming an arch, the diameter of which continued to extend for five minutes; at the end of eight minutes the arch had contracted owing apparently to small clots in the margin of the orifice. Dr. Dowler estimates the amount of blood which flowed out of the jugular vein at five pounds eight ounces. As the blood letting progressed the congestion and discoloration of the skin of the face diminished. Dr. Dowler concludes from this experiment that the circulation in the veins was more active than in health.

Case 9.—Yellow fever, intermittent type at its inception, with prolonged coma at its conclusion. Post-mortem heat August 29 1843, air about 83 degrees. S. F., born in Cincinnati; aged 24; sick eight days, (incipient coma); hand 104 degrees; axilla 105. The coma continued until death, three days after. The experiments with one instrument began thirty minutes after death, and continued about two hours. in the order of time as follows, having been recorded at intervals of two, three, five and sometimes more minutes: Axilla, 104 degrees, 105, 106, 106; thigh, 106, 107, 108, 107; axilla 107; left hypochondriac, 109, 109; right 109; hypogastrium 106; thigh 106.5, 106 5; lungs 102; heart 103; thigh an hour after the removal of all the abdominal viscera 106.5, and falling. Weather rainy; room 84. Body exposed to a free ventilation.

Case 10.—Yellow fever, post-mortem heat. Sept. 15, 1843; noon, air 89.5 degrees, A. G. G., born in New York; sick four days; hand 91 degrees; axilla 100; I bled to syncope, and cupped twice yesterday. Died at 1 P.M. In five minutes after death the experiments began, and were noted consecutively every five minutes. more or less: Axilla 102 degrees, 104, 106.5, 107, 106; thigh 106.5, 107, 106.5; axilla 105.5; thigh 106; left lung 106; upper lobe 103.5; heart 104.5; hypogastrium 106.5, 106.5; left hypochondriac 105.5; right thigh 106; left 106; axilla 104; left hypochondriac 104; hypogastrium 106.5; right hypochondriac 106.5; thigh 106.5; hypogastrium 106.5; left hypochondriac 104; right 104; rectum 105; hypogastrium 105. September 16, dead twenty-two hours; room 90 (noon); thigh 89; left lung 92; epigastrium 92. Incipient putrefaction.

Case 11.—Yellow fever, post-mortem heat. August 22, 1843, room 86. J. H., born in Virginia, aged 30; sick twelve hours, bled twenty ounces; the blood falling on the thermometer placed in a basin, gave in ten minutes 100 degrees; thirty minutes later, hand 103; both axillæ 107; (blood letting repeated) to syncope; cupping same day. August 23, cupped twenty ounces. Died at 10:25 A.M. Experiments began in ten minutes, ending at one hour and a quarter after death, in the following order: Axilla 100 degrees, 103; perineum 101; axilla 101.5, 101; rectum 103, 103; axilla 103; when the experiments ceased.

Case 12.—Yellow fever, post-mortem heat. August 15, 1843. Room 86. P. L., aged 20; sick three days; hand 99 degrees; axilla 104. August 16, hand 98; axilla 103. August 18, axilla 99. August 19, room 90; hand 98; axilla 101. August 20, died.

The observations beginning twenty-five minutes after death, were recorded about every five minutes continuously for three hours and forty-five minutes, with one thermometer, in the order following: Room 86 degrees. Axilla 100, 105, 106.5, 106.5, 106.5, 106, 106, 106.5; groin 104, 103.5; right hypochondriac 109; left 108.5; right iliac 108; left 108; right hypochondriac 108; thigh 109, 109; cardia of the stomach 109, 109, 109; circumference of the left lung 107; mediasternum 107, thigh 107, 107; right hypochondriac 107; left

107; heart 107; hypogastrium 107; thigh 107; heart 107; thigh 108, when the experiment ceased.

Case 13.—Yellow fever. Extraordinary heat during life, post-mortem heat. August 23, 1847. J. F., Irishman; aged 28; resident nine months; dying; experiments before death for forty-five minutes; hand 107 degrees; axilla at intervals during thirty minutes 110, 110.5, 110, 110. Died. Axilla at intervals during thirty minutes after death, 110.5; bend of the arm at ten minutes 107. Carried to the dead house, stripped and laid out. Axilla about one hour after death 108; at two hours 107¾. Darkness put an end to further experiments.

Case 14.—Yellow fever, post-mortem heat. August 28, 1848. Air about 89 degrees. N. L., born in France, aged 23; resident eighteen months. Dying; the hand and the ball of the thumb 94 degrees; bend of the arm 102, and falling; axilla 106. Dead house 89. The experiments began soon after death and continued with intervals for two and one-half hours, in the order following: Bend of arm 104 and falling; calves of the legs brought in contact 100; axilla 107; bend of the arm fifteen minutes 105.5. Stationary; axilla 107, soon 108, continuing for forty-five minutes, then declined to 107¾, and in one hour after to 103¾.

Case 15.—Yellow fever, post-mortem heat. August 12, 1847. H. M., a German, aged 45; hand 89 degrees; bend of the arm 93. Died one hour after. At one hour after death, the experiments were made for a few minutes, having been interrupted by night: Axilla 106, 106¼, and rising, being thirteen and one-sixth beyond the living state one hour before death.

Case 16.—Yellow fever, post-mortem heat. August 13, 1843. Air 91 degrees. R. C., born in Kentucky; aged 25; sick nine days; hand 98 degrees; axilla 102. Died next day. Room 93. The experiments began thirty minutes after death, and were noted every ten to twenty minutes ending with the seventh hour in the following order: Axilla 106.5; fold of the groin, 109; rectum 111, 111; axilla 104, 104, 104, 104, 104; thighs (in contact) 102; rectum 109, 109, 109; axilla after two hours exposure 100; rectum seven hours after death 102.

Case 17.—Yellow fever, post-mortem heat. Sept. 7, 1843. Air 80 to 83 degrees. J. G., aged 32; born in Ireland; sick five days; hand 98 degrees; axilla 102. September 11, hand 99; axilla 104. Died twenty-four hours after. The experiments began ten minutes after death; the body exposed to free ventilation; the room 90 degrees; the axilla ten minutes 109; left thigh five minutes 113; epigastrium five minutes 112; thigh old incision 109; epigastrium 112; right thigh new incision 111. After an interval of one hour the heart gave in eight minutes 109; the thigh in five minutes 109. After the removal of all the abdominal viscera the thigh still gave

109 degrees at about three hours after death. About one hour after death, both the arms including the scapulæ and shoulder-joints were amputated for the purpose of experimenting on the contractility of the separated limbs. The limbs and body together discharged from three to four pounds of blood, which did not seem to diminish the heat of the trunk, nor that of the separated limbs, so long as the experiments lasted.

Case 18.—Yellow fever, post-mortem heat. August, 1893; air of the room 84 degrees. L. L., aged 25; sick five days (cold for two days before death as in congestion), dead twenty minutes; weather very breezy; body resting on stone floor, freely ventilated; experiments with Reaumur's thermometer (now expressed by Fah.); lasted three hours and forty minutes, that is four hours after death, having been noted every five or ten minutes in the order of time as follows: Axilla 108 degrees: mouth 103; epigast. 108; perineum, without incision, 104; center of the thigh, 108½; mouth, 99¾; epigast. 108; thigh 106; axilla 104; epigast. 107 and 106½, 106½, 106½. Experiments now ceased for one hour and thirty-five minutes, during which time the mercury had fallen 5¾ degrees. It rained also. Epigast. air 104; circumference of the lungs (pluræ) 103; concave surface of the liver and base of the right lung 104; hypogastrium 103; concave surface of the liver and base of right lung 104, when the experiments ceased.

Case 19.—Yellow fever, post-mortem heat. Sept. 13, 1894; air of the room 86 degrees. N. L., a Frenchman, aged 58; sick eleven days; dead five minutes; experiments noted consecutively about once in five minutes; axilla 102; knees brought into contact 102; rectum 104: axilla after free exposure 104; thigh 105, 107, 108; epigastrium 107: thigh 108, 108, 104, 104; left lung 104; right 103.5; thigh 104, 104, 104; right lung (base) 103, left 102; thigh 103; epigastrium 103. September 14, seventeen hours after death, room 86; epigastrium 88.5; thigh 88 5; left chest, 88; calf of the leg 86; middle of the arm 88.5; epigastrium 88.5; thigh in one hour 86.

Case 20.—Yellow fever, post-mortem heat. Sept. 18, 1893; S. E., born in Germany; last from Texas; resident, one month; aged 23; sick five days; occasionally delirious; became comatose last night; now dying. Axilla 104 degrees; bend of the arm 102.5; palms 98.5. Died at sixteen minutes before 11 A.M. six minutes after death axilla 106, eight minutes 107, in two minutes later, 107; one minute 107¼; bend of the arm three minutes later (11 A.M.,) 104, one minute 104; palm two minutes 98.5. A few minutes past noon, resumed the experiments in the deadhouse, the temperature of which was about 88 degrees. The experiments were made successively in the following order, until 1 P.M.: Axilla 107 degrees, and stationary; epigastrium 107, 107.5; right and left hypochondriac 107.5, 107.5 ; center of upper third of thigh 107.5, 108 ; axilla 105 ; brain in the center 105 ; heart 107 ; brain 104¾, when owing to urgent circumstances the experiments ceased.

In this case during the agony, and even after respiration ceased, an universal tremor, with strong tension of the muscular system took place, while the fingers and forearms were found flexed, having been for a time very rigid. The treatment consisted of two doses of castor oil; a mixture of magnesia and charcoal in peppermint water, and at the close brandy.

An attentive study of the entire series of cases and observations recorded by Dr. Bennet Dowler establishes the following conclusions:

1. A temperature of 109 degrees, in the axilla in yellow fever is not necessarily fatal. It is probable that in such cases the temperature of the internal organs reaches 113 to 115 degrees.

2. Many cases of yellow fever, in the high temperature, and rapid progress and sudden termination resemble sunstroke (solar asphyxia, *coup de soleil insolatio*, heat stroke). When death takes place in the active stages of both diseases, the post-mortem heat may rise as high as 113 degrees, and remain at this point for varying periods of time.

3. In many cases of yellow fever, the rise of post-mortem temperature can not be referred solely to the arrest of respiration and of cutaneous and pulmonary transpiration and evaporation.

4. Post-mortem heat is not peculiar to yellow fever, but may occur after death from various causes, as lesions of the nervous system, traumatic tetanus, cholera, typhus and typhoid fevers, pneumonia and phthisis. The phenomena therefore can not be wholly referred to the action of the specific poison of yellow fever.

5. In many cases of yellow fever, as well as of other diseases, there is no post-mortem development of heat above that which existed at the moment of death; but on the contrary the bodies obey the ordinary

physical laws of the gradual conduction, radiation and loss of heat from the circumference to the center.

6. Post-mortem heat may be due to several causes, among which may be enumerated the following: *a*, chemic changes in the blood and organs, which continue for a certain period after death; and as the evaporation from the surface of the lungs and skin, as well as the refrigeration of the process of respiration have ceased the full effects of these chemic changes are manifested in the increased development of heat; *b*, the transmutation of the electrical forces and the modifications of physical force known as the muscular and nervous forces into heat.

CHAPTER V.

RELATIONS OF YELLOW FEVER TO SUNSTROKE.

The severer grades of yellow fever, attended with high temperature, reaching 108 degrees F., and over, and running a rapid course in two or three days to a fatal termination, present some symptoms in common with those cases of sunstroke in which the muscular and nervous prostration is accompanied with rapid and decided elevation of temperature. In cases of sunstroke, which occurred during the month of July, 1873, the temperature reached from 110 to 112 degrees F. in the axilla, at the time of death, and probably reached 115 degrees F. in the heart and internal organs. Similar elevations of temperature in sunstroke have been recorded by Dr. Bennet Dowler and others.

The phenomena of sunstroke appear to be dependent mainly upon the complex and unstable chemic constitution of the blood, and the dependence of the development of the physico-vital phenomena of the nervous and muscular forces, upon a definite degree of temperature. In common with other complex and highly elaborated organic fluids, which are continuously supplied with oxygen, the definite physical and chemical constitution of the blood can be maintained only within certain degrees of temperature. Unless a definite physical and chemical constitution of the blood be maintained, the necessary nutritive elements will not be supplied to the organs and tissues, and alterations in the secretions, and aberration of the muscular and nervous forces are inevitable. When the temperature of the blood rises above a certain point it is probable that a new series of chemic actions are developed; and compounds are generated which act as poisons to the nervous system. The rise of the temperature in the blood must also be attended with disturbances in the normal electric currents of the nerves and muscles. The great increase in the amount of carbonic acid, and the corresponding rapid consumption of oxygen in the blood must also be considered as important factors in the production of that state known as sunstroke. It is probable also, that as in certain cases of yellow fever, the center of the cerebro-spinal system which regulates animal temperature, as well as the vasomotor centers are involved. The derangement of these nervous centers is probably secondary to the chemical and physical alterations of the blood. It appears also, that the sudden failure of the heart, when the temperature of the blood rises above 110 degrees F., is due to the physical and chemical alteration and coagulation of the contractile constituents of the muscular fiber of the heart.

The remarkable phenomena of "sunstroke," "heat stroke" or "thermic fever," are most generally manifested in individuals working in intensely hot weather in ill ventilated apartments and cellars, in rooms artificially heated as in laundries, bar-rooms, wine cellars, and densely crowded tenement houses; in those addicted to the intemperate use of ardent spirits, in whom the capillaries of the brain are dilated and degenerated and the constitution of the blood deranged by the impairment of the functions of certain organs, as the liver and kidneys; and in teething children, exhausted by diarrheal discharges, and by the deranged reflex nervous actions, excited by the continuous irritation of the gums and dental nerves.

The foul air, loaded with the exhalations from the skin and lungs, and with carbonic acid, of the crowded houses of the poor, in narrow filthy streets, without doubt depresses the nervous and muscular forces, and prevents the proper maintenance of a definite temperature, by a free transpiration from the cutaneous and pulmonary surfaces.

The phenomena of sunstroke furnish conclusive evidence that mere elevation of temperature, apart from the action of any distinct febrile poison, may be the source of important changes in the blood, and fatal depression in the muscular and nervous systems. The phenomena of yellow fever and of other febrile diseases are rendered more complicated by reason of the disturbing agencies of the heat developed, and it is important that a thorough analysis of the blood, excretions and secretions in sunstroke be made, as forming a basis for the correct appreciation of the mere effects of temperature upon the blood and upon the nervous and muscular systems in yellow fever, independent altogether of the direct action of the febrile poison. Unfortunately, medical science is as yet without the necessary data for the institution of such an inquiry.

RESPIRATION IN YELLOW FEVER.

In many cases of yellow fever, the respiration is accelerated to a comparatively small extent; in cases of great severity, however, the respiration may be greatly increased in frequency, while the pulse may be even below the normal standard, and far below what is usual in other diseases with a similar elevation of temperature. The following cases present striking illustrations of the want of correspondence between the respiration, pulse and temperature in severe and fatal cases of yellow fever:

Case 1.—Yellow fever, black vomit, urinary suppression and death. G. H., age 33, laborer; native of Germany. Came on steamboat from St. Louis and had been in New Orleans one week before he was attacked. Entered hospital Oct. 11, 1873, at 4 P.M. Was vomiting black vomit when he entered. Sick five days before he entered the hospital, and in a state of nervous and muscular prostration and great capillary congestion. Black vomit abundant; gulps it up apparently without effort and squirts it on the wall and over the bed clothes. At 6 P.M. pulse 81, respiration 48, temperature of axilla 102.8. Complete suppression of urine, pain in epigastrium, delirious. Died next morning at 6 o'clock, fourteen hours after entering the hospital.

Case 2.—Yellow fever, jaundice, suppression of urine, black vomit, slow pulse, rapid respiration, death. J. A. H., age 30, native of United States, actor. Came down the Mississippi River on steamboat from St. Louis two weeks before his attack of yellow fever. Entered Charity Hospital Oct. 15, 1873, 5 P.M. Comatose; entire surface of a deep golden color; great congestion of peripheral blood vessels and capillaries; face of a purplish mottled hue; conjunctiva yellow and congested; pupils greatly dilated and incapable of responding to the stimulus of light; pulse small and thready. Threw up black vomit in the wagon before entering the hospital; moustache and side of face smeared with black vomit. Oct. 16, 1873, 8 A.M., pulse 110; respiration 22; temperature of axilla, 102 degrees: 8 P.M., pulse 68; respiration 68; temperature 105.5 degrees F. Has passed no urine since entrance into the hospital. With the catheter, I drew off about one fluid drachm of yellow urine, loaded with albumen, bile and granular casts of tubuli uriniferi and excretory cells of kidney. Upon standing, the urine let fall a heavy deposit of urates. The patient died three hours after this observation, at 11 P.M.

It is worthy of note that in this case with a temperature of 105.5 degrees and respiration 68 to the minute, the pulse was only 68 beats to the minute.

I have observed a similar condition, viz., the pulse and respiration of equal rapidity in no case of acute disease; and in fact while the pulse may be greatly reduced in frequency when jaundice supervenes in malarial fever and in pneumonia, and while in many diseases there is no absolute correspondence between the temperature and rapidity of the heart's action within narrow limits; at the same time yellow fever is distinguished from other acute diseases by the frequency with which the pulse diminishes in rapidity after the establishment of the febrile excitement. This proposition will be established by a comparison of the pulse, respiration and temperature in various diseases with the observations already recorded with reference to yellow fever.

In addition to the large number of cases of various diseases, recorded in the first volume of my "Medical and Surgical Memoirs," the following cases are presented for immediate comparison and definite conclusions:

Case 1.—Erysipelas of head and face engrafted on malarial fever. Patrick O'Hern; native of Ireland; weight 160 pounds; height five feet eight inches; has been in the United States three years; admitted into Charity Hospital, Nov. 22, 1876, with intermittent fever, contracted several months before, in the swamps of Arkansas. The intermittent fever was arrested by the usual remedies. On December 8, the patient was seized with a chill followed by high fever, which continued without remission and on the 9th erysipelatous inflammation of the face appeared. The features became rapidly swollen and the inflammation which began on the left cheek rapidly invaded the integuments of the face and head. The bowels were freely opened by a mercurial purgative and quinin administered. On the 12th, the patient was placed upon the following:

℞ Quinia sulph . . . . ʒi
Potassa chloratis . . . . ʒij
Acidi hydrochlorici dil. . . . . ʒiij
Zinc ferri muriates . . . . ʒiv
Aquæ menth. pip. . . . . f.℥ij
Mix.

Sig.: 30 drops in a wineglassful of water every four hours.

The local treatment consisted in painting the skin of the face and head, in advance of the line of inflammation with tincture of iodin. These measures finally arrested the progress of the inflammation and on December 20 the temperature of the axilla had fallen to 99 degrees F. The following table presents the relations of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.	
1876	M.	E.	M.	E.	M.	E.
Dec. 10	. . . .	116	. . . .	28	. . . .	104 °
" 11	100	114	24	22	103.5°	104.5
" 12	104	112	28	24	104	105
" 13	100	108	24	36	103	103.5
" 14	112	112	28	33	103	104
" 15	104	92	28	32	101.5	103
" 16	92	96	28	32	101	102
" 17	90	108	24	34	100	101
" 18	96	100	28	36	99	102.5
" 19	80	96	24	26	98	101
" 20	84	80	27	32	99	99
" 21	80	80	24	28	98	98
" 22	88	80	27	24	99.5	98
" 23	80	68	28	28	98	99
" 24	68	76	24	28	98.5	100
" 25	68	80	28	24	98.5	99
" 26	88	80	24	24	99	98.5
" 27	76	. . . .	24	. . . .	99	. . . .
" 28	63	66	24	20	99	99.5
" 29	72	80	24	28	99	98.5
" 30	80	96	28	28	99	. . . .
" 31	76	. . . .	20	. . . .	97.5	. . . .

The recovery of this patient was satisfactory and complete and he was discharged in good health, capable of performing manual labor.

Case 2.—Erysipelas of face and head. John Toole; laborer; age 50 years; weight 180 pounds; height five feet ten inches; dark hair, dark eyes, dark complexion; admitted to Charity Hospital Dec. 23, 1876. Integuments of face bright red and greatly swollen. The swelling is so great as to completely close the eyes and to obliterate the features. Previous to coming to New Orleans had worked on the levees, near Donaldsonville. Has a scar on the left side of the head caused by a blow about three weeks before. This injury appeared to be the exciting cause of the erysipelatous inflammation. The treatment of this case was similar to that instituted in the preceding case, and with like favorable results. The inflammation gradually subsided and after the complete restoration of health the patient was discharged. The following is the record of the pulse, respiration and temperature:

Date,	Pulse.		Resp.		Temp.	
1870	M.	E.	M.	E.	M.	E.
Dec. 23	80	76	16	24	98 °	100.5°
“ 24	80	80	20	24	98.5	100
“ 25	72	72	16	16	96	95
“ 26	56	68	16	16	94	96
“ 27	58	. . . .	16	. . . .	96.5	. . . .
“ 28	65	54	16	16	98.5	98.5
“ 29	64	60	13	12	99	97

Case 3.—Rubeola, dark purple eruption, high temperature, death. John Chilton; native of New Orleans; age 17; laborer; height five feet; weight 100 pounds; dark brown hair, gray eyes, dark complexion, sharp features; entered Charity Hospital Dec. 20, 1876. Patient states that he had been seized on the morning of the 9th inst. with a severe rigor, followed by intense fever, accompanied by pain in the head and back. December 11, 8 A.M., I saw the patient for the first time; face and surface generally greatly congested, presenting a purplish mottled appearance. Pulse 155; respiration 40; temperature of axilla 105 degrees F. Left lung congested; dull upon percussion, with physical signs of pleuro-pneumonia. Painful cough and respiration. Evening, rash over entire surface of a deeper color and of more distinct appearance; left lung and lower lobe of right lung congested. Pain, cough and jerking labored respiration. 8 P.M., pulse 156; respiration 47; temperature 106 degrees. Alcoholic stimulants, quinin and nutritious diet were ordered, but appeared to produce no beneficial results; neither was the local application of turpentine in

the form of stupes attended with any diminution in the severity of the pulmonary symptoms.

December 12, A.M., pulse 142, respiration 39, temperature 104.5 degrees F.; December 12, P.M., pulse 129, respiration 42, temperature 105 degrees F.; December 13, A.M., pulse 124; respiration 35; temperature 104.5 degrees F.; December 13, P.M., pulse 136, respiration 36, temperature 105 degrees F.; December 14, A.M., pulse 144, respiration 36, temperature 104 5 degrees F.

On the morning of December 14, the patient presented a livid appearance; great difficulty in breathing, dark blotches on his body running together, but more defined on arms, legs and feet. Tongue, which had been heavily coated, peeling off, leaving a raw surface. Intense thirst, feeble fluttering pulse. Died at 10 P.M.

Case 4.—Intermittent fever, congestion of right lung. K. Orbison; age 26 years; height five feet eight inches; light hair and blue eyes; native of Chicago. During the past summer was exposed to malaria in Mississippi and entered the Vicksburg hospital with chills and fever. Entered Charity Hospital on December 27, with high fever, furred tongue and congestion of lower lobe of right lung. The internal administration of quinin and Dover's powder (2 grains of each every three hours) and of the tincture of yellow jessamine (10 drops every three hours) and the local application of turpentine over affected lung, were attended by the resolution and disappearance of the pulmonary congestion. On January 5, the patient suffered with another paroxysm of fever which yielded readily to quinin. The following observations were recorded:

December 27, pulse 120, respiration 30, temperature 104 degrees F.; December 28, pulse 112, respiration 28, temperature 98 degrees F.; December 29, pulse 94, respiration 28, temperature 100 degrees F.; December 30, pulse 78, respiration 27, temperature 99 degrees F.; December 31, pulse 80, respiration 24, temperature 98 degrees F.; January 1, 1877, pulse 72, respiration 22, temperature 98 degrees F.; January 2, pulse 70, respiration 24, temperature 98; January 3, pulse 90, respiration 30, temperature 99 degrees F.; January 4, pulse 72, respiration 24, temperature 99 degrees F.; January 5, pulse 130, respiration 28, temperature 105.5 F.

Case 5.—Chronic malarial poisoning, general anasarca, ascites. Edwin Toll; age 39; admitted to Charity Hospital March 3, 1877. Native of Philadelphia; mechanic. Has been in Louisiana twelve months and has been living in the swamps of Quehito Parish. With the exception of syphilis twelve years ago, has enjoyed good health, up to August 1, 1876, when he was attacked with chills and fever, "swamp fever." The chills have continued at intervals up to present time. Pale, anemic, with greenish-yellow tinge of surface.

Spleen enlarged and painful upon pressure. Abdomen distended with liquid. Extremities, especially the legs and thighs greatly swollen and pitting deeply upon pressure. Urine high colored, moderately abundant, but free from albumen. It is probable that the liver has been deranged by the action of malaria, as well as by alcoholic stimulants, which he is said to have used in excess. Much benefit was derived from the following combination:

℞ Quinia sulph . . . . ℈i
Pulv. digitalis. . . . .
Pulv. scilla . . . . āā grs. v
Extract rhei. . . . .
Extract aloes . . . .
Extract colocynth comp. . . . .
Gamboge . . . . āā grs. x
Mix.

Divide into ten pills; one pill every six hours.

The action of the kidneys was promoted by cream of tartar in drachm doses, three times a day. After the bowels were freely moved by the preceding measures, the tincture of bark and quassia, together with quinin and iron were freely used and with marked benefit. Under these measures the dropsical effusion disappeared and the complexion assumed a healthy appearance. The following observations were recorded:

Date.	Pulse.		Resp.		Temp.	
1877	M.	E.	M.	E.	M.	E.
March 5	92	93	20	22	98°	100°
" 6	78	90	18	24	99	99
" 7	80	86	22	22	98	99.2
" 8	86	85	22	22	98.2	99
" 9	84	86	22	23	98.2	99
" 10	85	86	23	24	101	100
" 11	76	78	22	22	99	99
" 12	80	. . . .	23	. . . .	98.2	. . . .
" 13	82	. . . .	23	. . . .	98.2	. . . .

Case 6.—Acute mania; general paralysis of lower extremities. Patrick J. Fitzpatrick; age 40; native of Ireland. Has resided in New Orleans thirty years. Admitted to Charity Hospital, Feb. 20, 1877. Can give no coherent account of himself. Laughs, cries, screams, crows like a cock, preaches and talks wildly and incoherently. Religion and military affairs appear to occupy his mind chiefly. Ofttimes repeats the prescriptions, as I give them to the student, passing from bed to bed; hearing appears to be very acute. Does not rest at night; can not stand on feet but falls forward. Blisters to back of neck and bromid of potassium and hydrate of chloral, as well as minute attention to the

condition of the bowels appeared to afford some relief and to moderate the ravings to some extent. The case, however, appeared to be but little improved when I gave up the case on March 15.

The following observations were recorded: March 8, pulse 100, respiration 24, temperature 99 degrees; March 9, pulse 100, respiration 25, temperature 99 degrees; March 10, pulse 98, respiration 24, temperature 99 degrees; March 11, pulse 80, respiration 24, temperature 99 degrees; March 12, pulse 85, temperature 98.2 degrees; March 13, pulse 85, respiration 24, temperature 98.2; March 14, pulse 88, respiration 24, temperature 99 degrees.

Case 7.—Double pneumonia supervening in malarial fever. James Rooney; native of Ireland; age 30 years; light complexion; weight in health 165 pounds; height five feet eight inches. Stone cutter by trade; has been in America four years, but has not worked at his trade during this time. In November, 1876, he obtained work on the Jackson railroad, and labored in mud and water up to his knees. Two weeks after beginning work in the swamps, was seized with chills and fever, and returned to New Orleans. Was sick one week before entering the hospital and during this period had little or no attention. Admitted to Charity Hospital, Jan. 24, 1877. I saw him for the first time on the 25th; found upon examination that the entire right lung and the lower portion of the left lung were completely solidified. All respiratory sounds with the exception of the tubular breathing were absent from the right lung. Expectoration very scant; respiration hurried; great congestion of face, with purple lips and tongue; rapid pulse and high temperature; absence of chlorids from the urine.

Notwithstanding the unfavorable and almost hopeless aspects of this case, the persistent use of quinin, Dover's powder, and alcoholic stimulants, carbonate of ammonia and nutritious diets and the local application of turpentine, were attended with marked improvement of the symptoms. The chlorids returned to the urine, the left lung was restored and portions of the right lung manifested crepitant, subcrepitant and mucous râles. A large cavity found in the superior and middle lobes of the right lung. During the breaking down of the lung tissue, the patient suffered from hectic fever, profuse sweats and great exhaustion. Convalescence although slow was finally and completely established.

The following observations will illustrate the relations of the pulse, respiration and temperature in this grave case:

Date.	Pulse.		Resp.		Temp.	
1877	M.	E.	M.	E.	M.	E.
Jan. 25	120	105	36	28	104 °	103.5°
" 26	112	108	28	32	101	101.5
" 27	104	108	28	28	101	102
" 28	100	100	28	32	100	103
" 29	96	100	32	28	100	101
" 30	96	100	28	32	100.5	102
" 31	84	92	28	24	100	100
Feb. 1	84	80	28	28	100.5	100.5
" 2	96	92	28	28	99.5	101
" 3	76	92	28	32	100	101
" 4	84	. . . .	36	. . . .	100	. . . .
" 5	84	96	24	28	100	101
" 6	96	108	28	28	100.5	101.5
" 7	104	108	28	32	100	101.5
" 8	104	112	28	36	100	101
" 9	104	104	24	28	100	101
" 10	92	. . . .	28	. . . .	99	. . . .
" 11	. . . .	120	. . . .	28	. . . .	102
" 12	108	112	28	24	100	101.5
" 13	100	. . . .	32	. . . .	100	. . . .
" 14	112	120	28	24	99.5	101.5
" 15	120	116	28	25	100	101
" 16	100	100	28	32	100	99
" 17	100	. . . .	26	. . . .	99.5	. . . .

Case 8.—Double pneumonia, bloody sputa, recovery. Robert Hanley; Irishman; came to America in 1846 and has been living in New Orleans since 1847. Weighs 150 pounds; florid complexion, light hair and blue eyes. On Jan. 6, 1877, while working in the hold of a ship, which was very close and hot, was thrown into a great heat and profuse perspiration. Being called suddenly, he left the ship without his coat (his shirt being saturated with perspiration), went on shore and was exposed to a cold wind, and in a few minutes was seized with a severe pain in the left side below the seventh rib. The pain continued to increase on the seventh and eighth, and upon the last day he began to cough and to expectorate thick, bright red sputa. Could not lie on the right side in consequence of the pain and oppression of breathing. On the tenth, eleventh and twelfth the pain and oppression of breathing increased and was attended with high fever. Admitted to Charity Hospital January 12. January 13, A.M., tongue red at tip and edges and furred in the center; sputa thick, brownish-red and streaked with bright blood; high fever, anemia, restless. Respiration labored and attended with sharp pain greatest over region of middle and lower lobes of left lung. Face flushed. Skin bathed in hot perspiration. Upon percussion, dullness amounting to almost complete flatness over middle and lower lobe of left

lung and over lower lobe of right lung. Auscultation revealed tubular breathing and crepitant râles upon full inspiration over the regions indicated. The patient was placed upon 2 grains each of quinin and Dover's powder every three hours, and five drops of the tincture of yellow jessamine every two hours. Turpentine stupes were applied over the affected portions of both lungs; milk punch and beef tea ordered in small quantities at regular intervals of two hours. This treatment was continued for several days and the bowels were opened when necessary by the ordinary efferverscing powders. On the 20th there was a marked improvement and the chlorids which had been absent reappeared in the urine and the sputa contained much less blood. Convalescence continued and on the 25th the patient was able to walk about the ward. He was finally restored to health, although he was allowed to remain in the ward for four weeks, until his strength was fully restored. The following table presents the relations of the pulse, respiration and temperature:

Date.	Pulse.		Resp.		Temp.	
1877	M.	E.	M.	E.	M.	E.
Jan. 13	109	118	42	40	101.5°	103.2°
“ 14	114	104	41	41	103	101.5
“ 15	90	100	36	38	101	104
“ 16	86	87	30	37	100	101.5
“ 17	88	89	36	37	100	99
“ 18	72	76	29	32	97	97
“ 19	68	67	28	32	98	98.3
“ 20	96	65	33	28	98.3	98.3
“ 21	94	. . . .	32	. . . .	98.5	. . . .
22	. . . .	72	. . . .	24	. . . .	97
23	61	62	22	26	97	97.8
24	65	60	22	20	97.5	98
25	60	. . . .	20	. . . .	97	. . . .
26	82	. . . .	24	. . . .	98.5	. . . .

Case 9.—Pneumonia; middle and lower lobes of left lung involved. John Dugan; age 21; native of New Orleans; longshoreman. Has enjoyed good health up to July, 1876, when he suffered with chills and fever. On Dec. 15, 1876, admitted to Charity Hospital with pneumonia and was discharged Jan. 6, 1877. February 21, seized with severe chill, attended with cough, high fever and severe pain in left side. Admitted to Charity Hospital February 25; high fever, rapid pulse and respiration; tongue red at tip and edges and furred in center; middle and lower lobes of left lung dull upon percussion, with tubular breathing and crepitant râles; chlorids absent from urine; sputa thick and rusty colored. The treatment was similar to that instituted in the preceding case; namely, quinin and Dover's powder, tincture of yellow jessamine, turpentine stupes, milk punch and beef tea. The

results were favorable. February 27, oppression of breathing and pain in left side diminished; February 28, free from pain, although left side still dull upon percussion; sputa still mixed with blood; small quantity of chlorids in urine. Continued to improve up to March 3, the expectoration being quite copious, with progressive diminution of dullness; reappearance of crepitant, subcrepitant and mucous râles over the affected portions of the left lung. March 3, the temperature and pulse were normal and patient able to sit up with good appetite. Recovery rapid and complete. The following observations were recorded:

Date.	Pulse..		Resp.		Temp.	
1877	M.	E.	M.	E.	M.	E.
Feb. 26	120	108	40	36	103 °	102 °
" 27	94	105	28	44	99	101
" 28	80	86	28	37	98	101
Mch. 1	78	77	26	28	98.2	99
" 2	72	76	22	28	98	98.2
" 3	72	73	24	24	98	98.2

It is evident that in the preceding cases of pneumonia, the combined use of quinin, Dover's powder and tincture of yellow jessamine reduced the frequency of the pulse, diminished the temperature and promoted the arrest and resolution of the pneumonic inflammation.

Case 10.—Pneumonia of right lung, engrafted on malarial fever. John C. Keller; age 24 years; admitted to Charity Hospital Feb. 4, 1874. Occupation, clerk. Seized with a severe chill January 30, followed by high fever. Middle lobe of right lung in second stage of pneumonic inflammation; lower lobe of left lung in similar condition. February 4, evening temperature of axilla 104.8 degrees. February 5, morning, respiration 36, pulse 126, temperature 103; evening temperature 105. Suffers from a painful cough and oppression of breathing; sleep disturbed; skin dry and hot; tongue red at tip and edges and coated heavily with yellow fur. Sputa abundant and mixed with considerable quantities of blood. February 6, morning, respiration 38, pulse 118, temperature 104.8; evening temperature 105.3. February 7, morning, respiration 39, pulse 128, temperature 104.1. This day the patient had a profuse hemorrhage from his lungs and his stools also contained blood. The hemorrhage was attended with a marked depression of temperature, as is often the case in the black vomit (hemorrhage from the stomach) in yellow fever. February 8, morning, respiration 37, pulse 121, temperature 103; evening temperature 101.5.

February 9, morning, respiration 38, pulse 125, temperature 101.8; evening temperature 101.6. February 10, morning, respiration 34, pulse 108, temperature 101.2; evening temperature 103.2. February 11, morning, respiration 34, pulse 104, temperature 102; evening temperature 102.6. February 12, respiration 30, pulse 101, temperature 101.5; evening temperature 101.3. February 13, morning, respiration 32, pulse 120, temperature 100; evening temperature 102. February 14, respiration 32, pulse 96, temperature 100. February 15, morning temperature 98.9; evening, 100.8. The patient continued to improve and was discharged March 3.

It is worthy of note that in this case of pneumonia, hemorrhage from the lungs was attended, as in cases of hemorrhage from the stomach and bowels in yellow fever, with a decided reduction of temperature, and also that the loss of blood from the lungs in this case appeared to be productive of good and to mark the period of convalescence.

Case 11.—Pleuro-pneumonia, solidification of entire lung, death. I. D., age 28; entered Charity Hospital Feb. 19, 1874. February 20, morning, respiration 36, pulse 108, temperature 102; evening temperature 102.7. February 21, respiration 26, pulse 95, temperature 103. February 22, respiration 39, pulse 104, temperature 102.7. February 23, respiration 39, pulse 115, temperature 103.5. February 24, respiration 40, pulse 120, temperature 102.5. February 25, respiration 38, pulse 114, temperature 97. This patient died on the night of the 25th. Entire right lung solidified, as shown by post-mortem examination. Great capillary congestion before death.

Case 12.—Abscess of lungs, following double pneumonia. A. R., age 26; admitted into Charity Hospital with pneumonia (double) Nov. 20, 1873. During the active stages of the pneumonic inflammation the temperature of the axilla reached 109 degrees F.; the pulse ranged from 100 to 130, and the respiration from 50 to 58 per minute. A large portion of the right lung passed into the stage of solidification and gray hepatization, and a large cavity formed in the middle lobe of the right lung; the patient was not restored to health until the end of four months. During the period in which these changes were taking place in the lungs, daily observations were made upon the pulse, respiration and temperature, numbering over seven hundred. During a period of one hundred and twenty days, while structural alterations were taking place in the lungs the pulse ranged from 80 to 130; the respiration from 22 to 40, and the temperature of the axilla in the morning from 99 to 102 degrees F., and in the evening from 100 to 104 degrees F.

Case 13.—Pneumonia engrafted on malarial fever. P. W., age 33; entered Charity Hospital Dec. 11, 1873. Had suffered with chills and fever for one week before entering the hospital. Lower lobes of both lungs involved on the 12th. During fourteen days the active stages of the disease, the morning temperatures ranged from 101.5 to 105 degrees, and the evening temperatures from 103 to 106 degrees. On the fifteenth day there was a distinct remission of the fever, which rose again on the seventeenth day, declined on the eighteenth to twenty-fourth days. On the twenty-fifth, the temperature again rose, apparently from the supervention of a fresh paroxysm and the involvement of other portions of the pulmonary tissues in the pneumonic inflammation. In this which was the last paroxysm and followed by convalescence the axillary temperature reached 104 degrees. The pulse in this case ranged from 72 to 120; and the respiration from 24 to 40 per minute.

Case 14.—Double pneumonia. Michael Castello; age 30; entered Charity Hospital Jan. 13, 1874. Had chills and fever two days before entering hospital. January 15, delirious. Region of left lung dull upon percussion, with crepitant and subcrepitant râles. Lower lobe of right lung dull upon percussion with crepitant râles, oppressed breathing with severe cutting pains. Tongue red at tip and edges and coated with yellowish fur in center. Urine high colored; specific gravity 1022; strong acid reaction, and mere trace of chlorids; no albumen. Morning, pulse 120, respiration 25, temperature 105.2 degrees; evening, temperature 105 degrees. January 16, delirious; increased dullness over left lung and lower lobe of right lung. with tubular breathing, bronchophony and increased vocal resonance. Specific gravity of urine 1025; high colored; strong acid reaction; absence of chlorids; no albumen. Morning, pulse 114, respiration 32, temperature 104 degrees; evening, temperature 105 degrees. January 17, morning, pulse 114, respiration 32, temperature 104 degrees; evening, 104.2. Trace of chlorids in urine. January 18, morning, pulse 124, respiration 40, temperature 103.5 degrees; evening, temperature 103.5 degrees. Trace of chlorids in urine. January 19, morning, pulse 114, respiration 32. temperature 101.5 degrees. Trace of chlorids in urin. There has been a marked decline of temperature, the pulse and respiration are also less frequent, and the delirium is disappearing. January 20, morning, pulse 98, respiration 26, temperature 99.5; evening 100.6 degrees. While the physical signs show no marked change, the urine shows a marked increase of the chlorids, the temperature has declined and the circulation and respiration are slower and more natural. The delirium has disappeared and the patient shows signs of marked improvement. January 21, continues to improve. Morning, pulse 84, respiration 28, temperature 98.5; evening, 99.8. Chlorids increasing in urine. January 22, morning, pulse 80, respiration 24, temperature 98.4. January 23, continues to improve; the air is again entering the solidified portions of the lungs and there is a great increase of the chlorids in the urine. Morning, pulse 66, respiration 24, temperature 97.5; evening, temperature 99.2. January 24, pulse 70, respiration 20, temperature 99.2; evening, temperature 100.3. Specific gravity of urine 1022 Chlorids abundant. January 25, morning, pulse 74, respiration 22, temperature 98.4; evening, temperature 99.6. January 26, morning, pulse 72, respiration 20, temperature 98; evening, 98.7. January 27, morning, pulse 76, respiration 20, temperature 98.4; evening, temperature 101.1. Continued to improve and on the 28th was able to dress and come down to amphitheater.

Case 15.—Pleuro-pneumonia of left lung, recovery. L. Winans; age 30; native of Ohio; on the trip from St. Louis to New Orleans was seized with severe chill followed by diarrhea and pain in left side. Entered Charity Hospital, ward 27, Jan. 3, 1874. Face flushed, frequent and painful cough, rusty colored pneumonic sputa, dullness over lower portion of left lung, bronchophony, bronchial respiration, increased local fremitus, vesicular murmur heard only in clavicular region of left lung, some pleuritic pain in left side, hot dry skin, dry red tongue, thirsty, loss of appetite and restlessness. Morning. pulse 110; respiration 28; temperature 104. The treatment which I described at length in the article on "Pneumonia," vol. I, "Medical and Surgical Memoirs," was instituted. Three and a half grains each of quinin and Dover's powder were administered every four hours, and 4 drops of the tincture of veratrum virid, every four hours, alternating with the powders of quinin and ipecac et opii, turpentine stupes applied over region of affected lungs. Beef tea and milk punch freely administered. Mere trace of chlorids in urine. January 4, morning, pulse 104, respiration 28, temperature 102. The quinin and Dover's powders and veratrum appear to have effected a decided reduction in the temperature, although patient is restless and anxious, and complains of some pain in the left side. January 5, morning, pulse 80, temperature 100.4, respiration 28. No chlorids in urine, specific gravity 1018, strong acid reaction, bowels moved, no albumen in urine. Evening, temperature 100.2. January 6, temperature still depressed, trace of chlorids in urine; substituted tincture of yellow jessamine 10 drops every four hours for the tincture of veratrum. Morning, pulse 94, respiration 32, temperature 102.5. Evening, temperature 105.5. January 7, left lung completely solidified, sputa abundant and rusty colored. Morning, pulse 112, respiration 32, temperature 104.4. Evening, temperature 103.5. January 8, pulse 100, respiration 36, temperature 102.5. Evening, temperature,103.5. January 9, morning, pulse 104, respiration 30, temperature 100.2. Evening, temperature 102. January 10, pulse 80, respiration 26, temperature 98.9. Evening, temperature 99. January 11. morning, pulse 80, respiration 26, temperature 98.2. Evening, temperature, 99. Chlorids reappear in urine, specific gravity 1018, no albumen has appeared in the urine up to the present time. January 12, morning, pulse 78, respiration 26, temperature 98.5. Evening, temperature 98.8. On this day chlorids reappeared in the urine, continued to improve and was discharged cured.

Case 16.—Pneumonia. Michael Burk; age 22; native of Germany; laborer. Entered Charity Hospital Jan. 12, 1874. Was taken with a chill followed by a high and continued fever four days before entering the hospital. January 13, great pain in left side, hot dry skin, dullness over left lung, bronchophony and increased local fremitus with tubular breathing. Morning, pulse 120, respiration 52, temperature 101.3. January 14, patient complains of great pain in left side and lower portion of right lung, which is dull upon percussion, rusty colored sputa. Morning, pulse 118, respiration 50, temperature 104.5. Quinin and Dover's powder, three grains of each every three hours administered internally. Tablespoonful of officinal solution of acetate of ammonia every four hours. Turpentine stupes to chest. Beef tea and milk punch. January 15, rested badly during night, delirious, tubular breathing and dullness upon percussion over left lung, dullness upon percussion over lower portion of right lung. Morning, pulse 112, respiration 56, temperature 102. January 16, very restless, delirious, physical signs the same, absence of chlorids in urine, no albumen in urine. Morning, pulse 104, respiration 48, temperature 102.6. January 17, delirious, necessary to confine patient to the bed, no chlorids in the urine, trace of albumen in urine. Morning, pulse 124, respiration 64, temperature 103.5. January 18, wild delirium, was held in bed all night by nurses, albumen in abundance in urine but no chlorids. Morning, pulse 120, respiration 64, temperature 103.5. Evening, temperature 105. January 18, patient more quiet, albumen not so abundant in urine, chlorids absent. Morning, pulse 132, respiration 62, temperature 104. Evening 104.3. January 20, patient in semi-conscious state, traces of albumen in urine, no chlorids. Morning, pulse 112, respiration 64, temperature 103. Evening 104.5. Alcoholic stimulants have been freely given. January 21, no delirium, rested well during the night, some appetite, albumen disappearing from the urine, chlorids returning, expectoration more free. Morning, pulse, 90, respiration 36, temperature 101.9. Evening, temperature 102. Convalescence was slow in this case. The patient sat up for the first time about February 2. The temperature remained elevated until February 10; it reached 103 on this and for a period of twenty-two days, from January 21 to February 10 ranged in the morning from 101 to 103, and in the evening from 101 to 104. Pulse from 90 to 140, and respiration from 32 to 58 per minute. Recovery tedious but satisfactory.

Case 17.—Double pneumonia, death. Peter Wonts, age 27. Entered Charity Hospital Dec. 18, 1874; pneumonic inflammation of lower lobes of both lungs, great oppression in breathing, purple lips and extremities, delirium. The morning temperatures ranged from 97.5 to 102; and the evening temperatures from 100 to 105.5, pulse from 96 to 120, respiration from 25 to 35. This patient died on the tenth day after entering the hospital.

Case 18.—Pleuritis. T. Lynch, age 21, native of Ireland; entered Charity Hospital Dec. 20, 1873. Had been treated in this institution two weeks before, for intermittent fever. December 20, evening. Intense pain in right side, dullness upon percussion of right lung with friction, pleuritic sounds in respiration. Pulse 130, respiration 25, temperature, axilla 105. December 21. Pain in side intense, morning pulse 77, respiration 23, temperature 101. Evening pulse 110, respiration 23, temperature 106. The patient has been treated with quinin and Dover's powders, 3½ grs. of last every three hours, and turpentine stupes to chest, these measures appeared to be beneficial and to rapidly reduce the temperature. December 22. Morning, pulse 76, respiration 30, temperature 99; evening, pulse 80, respiration 30, temperature 100. At 3 P.M. on the preceding day the patient had a severe chill followed by high fever in which the temperature reached 106. A large plaster was applied to the side, quinin freely administered with beneficial results as stated. December 23, slept better during the night, improving, pain in side much relieved. Morning, pulse 72, respiration 25, temperature 99; evening, pulse 80, respiration 28, temperature 101. December 24. Morning, pulse 86, respiration 32, temperature 100; evening, pulse 82, respiration 30, temperature 102. December 25. Morning, pulse 75, respiration 28, temperature 99.5; evening, pulse 83, respiration 30, temperature 101. December 26. Morning, pulse 70, respiration 20, temperature 99; evening, pulse 75, respiration 24, temperature 100. The patient continued to improve and was discharged in a few days after this observation.

Case 19.—Pneumonia. Supervening on malarial fever, cured. N. T,, aged 24, entered Charity Hospital Jan. 6, 1874. Arrived in New Orleans in destitute condition and slept on levee nights and January 5 had a violent chill followed by congestion of lower lobes of both lungs and high temperature, 106.2, pulse 120, respiration 38. Under the action of the quinin and Dover's powders, rapid reduction of temperature and improvement of symptoms.

Case 20.—Typhoid fever. T. P., entered Charity Hospital Jan. 11, 1874, rapid pulse, high temperature, great nervous prostration, muttering delirium. During a period of thirty-two days the temperature oscillated between 99 and 106. The temperature in the morning varied from 1 to 7 degrees lower than the evening temperature. On January 12, (second day after entrance) the morning temperature was 104 and the evening temperature 105. On January 23 the morning temperature was 99 and the evening 106; on the 28th morning temperature 100, evening 103; the 30th, morning temperature 102, evening 105. The pulse ranged from 90 to 110, respiration from 30 to 56.

Case 21.—Typhoid fever. H. F., age 27. Entered Charity Hospital November 29, high fever continued from November 29 to December 21. The range of the temperature and pulse are as follows: November 29, evening, pulse 102, temperature 105. 30th, morning, pulse 86, temperature 103; evening, pulse 94, temperature 105. December 1, morning, pulse 82, temperature 103; evening, pulse 90, temperature 105. 2d, morning, pulse 80, temperature 103; evening, pulse 82, temperature 103.5. 3d, morning, pulse 86, temperature 103.5; evening, pulse 90, temperature 104.5. 4th, morning, pulse 90, temperature 103; evening, pulse 86, temperature 104.5. 5th, morning, pulse 82, temperature 101; evening, pulse 88, temperature 103.4. 6th, morning, pulse 80, temperature 101; evening, pulse 88, temperature 103. 7th, morning, pulse 100, temperature 104; evening, pulse 94, temperature 104. 8th, morning, pulse 82, temperature 99; evening, pulse 104, temperature 105. 9th, morning, pulse 90, temperature 112.5; evening, pulse 88, temperature 104. 10th, morning, pulse 82, temperature 100.5; evening, pulse 104, temperature 104. 11th, morning, pulse 90, temperature 100.5; evening, pulse 88, temperature 104.5. 12th, morning, pulse 108, temperature 100; evening, pulse 94, temperature 104. 13th, morning, pulse 86, temperature 103; evening, pulse 80, temperature 101. 14th, morning, pulse 84, temperature 100; evening, pulse 80, temperature 100. 15th, morning, pulse 80, temperature 99.5; evening, pulse 80, temperature 100.3. 16th, morning, pulse 90, temperature 98.8; evening, pulse 80, temperature 100.2. 17th, morning, pulse 86, temperature 98.5; evening, pulse 80, temperature 100. 18th, morning, pulse 80, temperature 99.5; evening, pulse 120, temperature 103.6. 19th, morning, pulse 110, temperature 102; evening, pulse 100, temperature 103. 20th, morning, pulse 96, temperature 101.5; evening, pulse 92, temperature 100.5. After this date the temperature gradually descended to the normal state and the bowels became constipated.

Case 22.—Typhoid fever. John Martin, age 24, entered

Charity Hospital November 21. In this case the diarrhea and fever continued for a period of forty-four days. The morning temperature ranged during this period between 98 and 105, and the evening temperature between 98.5 and 105 degrees F. The pulse ranged from 70 to 120; the respirations from 24 to 34 per minute.

Case 23.—Albuminuria and malarial fever. J. P. Banem; age 30; admitted to Charity Hospital Jan. 20, 1874. Has been living on the banks of the Mississippi River, where he contracted intermittent fever, from which he has suffered during the past three months. The condition of the patient is as follows: Great anemia; tongue swollen, indented by the teeth along the edges, pale, flabby and tremulous; complexion sallow and of a waxlike greenish tinge; features swollen; abdomen distended with fluid; lower extremities swollen; complains of headache and dullness of intellect; urine high colored and scant, and loaded with albumen. This patient was kept under observation for about three months (ninety days), and at various periods the urine contained blood corpuscles in addition to the albumen and urinary casts. The treatment consisted mainly in keeping the bowels open by means of saline purgatives, the occasional use of quinin for several consecutive days, and the administration of diuretics and alteratives, among which may be mentioned iodid of potassium combined with wine of colchicum and syrup of squills. The strength was maintained by nutritious diet and the preparations of iron. Under these measures the improvement was gradual and decided; the dropsical effusion disappeared and the patient was discharged at his own request in April.

In this case the action of the heart was quick and irritable; no valvular disease, however, was detected. Edema of the lungs was a troublesome symptom. The pulse was rapid and irritable, varying from 100 to 130; the respiration was often embarrassed and ranged from 20 to 30; the temperature in the morning ranged from 97.5 to 100, and in the evening from 99 to 101. This case appears to have been dependent to a great extent upon the action of malaria.

Case 24.—Constitutional syphilis. R. N. W., age 49½ years. Entered Charity Hospital Dec. 12, 1873. Contracted syphilis in 1866; suffers with severe pains in the extremities and head which increase greatly during the night and destroy his rest. December 17, morning, temperature 99.6, pulse 80, respiration 20; evening, temperature 100.6. 18th, morning, temperature 98.5; pulse 80, respiration 18; evening, temperature 100. 19th, morning, temperature 98.5, pulse 96, respiration

24; evening, temperature 100. The observations upon the temperature and pulse were continued for a considerable length of time, and the former ranged from 98 Fah. in the morning to 101 in the evening and the latter from 80 to 100.

Case 25.—Cirrhosis of liver; ascites; dysentery and diarrhea. T. G., age 50; native of Cincinnati, Ohio. Entered Charity Hospital March 4, 1874. Has been a hard drinker for twenty-two years. Had malarial fever in July, 1873. Has lost flesh, his weight decreasing from 154 to 123 pounds. Abdomen distended with liquid; lower extremities edematous; bowels very loose. In this case the temperature in the axilla varied in the morning from 98.5 Fah. to 99.5; and in the evening from 99 to 100.5; the pulse varied from 70 to 90 per minute.

Case 26.—Aneurysm of the arch and descending portions of the aórta. L. D.; age 43; native of Ireland; has lived in America twenty years; served in the Confederate Army about twelve months. Laborer. Entered Charity Hospital March 4, 1874. States that about one month before entering the hospital, while rolling a bale of cotton, felt a sudden pain in the abdominal region and upon examination found a small tumor which has been steadily increasing from that date, and at times interferes with his respiration. The second sound of the heart is accompanied by a distinct aortic murmur and the pulse is irregular and at times dicrotic. The temperature in this case ranged from 98.4 Fah. to 101.3, and the pulse from 80 to 90.

Case 27.—Aneurysm of arch of aorta and phthisis pulmonalis. Nicholas Batterton; age 33 years. Admitted into Charity Hospital Dec. 1, 1873. Patient says that he has been sick for one year and that the disease began with pneumonia. His brother died with phthisis. After the attack of pneumonia, suffered with a troublesome cough, and has had hemorrhage from the lungs seven times. The arch of the aorta is dilated and the tumor is evident in the supra-clavicular region, where the pulsation can be seen and also readily felt. When the fingers are pressed above the superior border of the sternum, downward and backward, the round pulsating contour of the aneurysm is distinctly felt. Observations were continued in this case, for one hundred and twenty days, and during this period the pulse varied from 60 to 100, the respiration from 30 to 40 per minute, the temperature in the morning from 99 to 101, and in the evening from 100.3 to 103.5. Auscultation revealed a distinct diastolic mitral murmur, propagated along ascending aorta and along the arch. Upon cod-liver oil and syrup of the hypophosphites, this patient continued much in the same state, until the winter of 1874, when the emaciation became more rapid and the patient died Nov. 2, 1874. Death in this case was in no manner attributable to the aneurysm of the aorta.

Case 28.—Abscess of thigh and extensive bed sores resulting from yellow fever. E. Scheiss; age 37; native of Germany. Entered Charity Hospital Oct. 29, 1873. Had been sick with yellow fever several days before entrance. Attack severe attended with high fever, vomiting and intense jaundice. During the period of convalescence and calm, a swelling was observed in left leg which greatly increased. From the confinement and also from the deranged state of the fluids caused by the yellow fever poison, large bed sores formed upon sacrum and hips. A large abscess of the thigh was opened. Patient died from exhaustion induced by hectic fever February 24. During 140 days following the active stage of the yellow fever the temperature ranged from100 to 105.2; the evening temperature ranged from 101.5 to 105.2. The pulse varied from 100 to 140 and the respiration from 24 to 34.

Case 29.—Mitral regurgitation, hypertrophy and dilatation of heart, and Bright's disease. R. Manez; age 36. Admitted to Charity Hospital Jan. 29, 1874. Had yellow fever in 1854 and 1858; contracted syphilis in 1864; suffered with rheumatism in 1869; malarial fever in 1871 on Red River. In August, 1873, feet began to swell, the liver was enlarged and the action of the heart irregular. Urine loaded with albumen. Owing to the obstruction to the circulation the patient suffered with the symptoms of edema of the lungs, accompanied with bloody expectoration, ascites and anasarca. In this case the pulse varied from 52 to 120; respiration from 20 to 28; temperature from 98 to 102.2. The temperature attained the highest point in the evening of February 5. The reduction of the pulse to 52 beats per minute was due to the action of digitalis.

A comparison of the results recorded illustrating the changes of the pulse, respiration and temperature in various diseases with those of yellow fever, establishes the following conclusions:

1. Yellow fever is a distinct specific disease.
2. Yellow fever is distinguished from other diseases by the frequency of the occurrence of black vomit and jaundice, and the progressive diminution of the frequency of the pulse after the establishment of the maximum temperature, also the want of correspondence observed in many cases between the rapidity of the respiration and the action of the heart.
3. As a general rule in acute diseases, the rapidity of the action of the heart corresponds with the rapidity of the respiration; but in some cases of yellow

fever in which the respiration is increased fourfold, the pulse actually beats less rapidly than in health.

4. Yellow fever is a continued form of a single paroxysm, in which a powerful depressing effect is evidenced upon the action of the heart. The cause of the depressed action of the heart will be discussed in the following chapter relating to the changes of the blood.

5. If the thermometric changes of yellow fever be projected as in the form of charts, and if a comparison be instituted with the thermometric changes in other diseases, it will be observed that those of the former disease more nearly resemble the rapid rise and sudden fall of temperature observed in varioloid without secondary fever, mild scarlatina, and simple uncomplicated pneumonia which runs its course without fresh accessions of inflammatory action; while on the other hand, they differ materially from the rapid and oft recurring elevations and depressions of temperature characteristic of the various forms of paroxysmal malarial fever.

6. The cause of the rapid rise and declension of the temperature in yellow fever must be sought chiefly in the changes induced in the blood and in those organs upon which the circulation and integrity of the blood depends. Neither the rapid rise nor the sudden declension of the temperature can be referred wholly to the effects of the yellow fever poison upon the nervous system.

While it must be admitted that the experiments of Chossat, Sir Richard Brodie, H. Nasse, Claude Bernard, Brown-Séquard, Budgé, Waller, De Ruyter, Schiff, Naunyn and Quincke, and the experiments of Tscheshichim, in which division of the medulla oblongata near its junction with the pons, caused a remarkable elevation of temperature from 102.92 to 108.68 Fah. and finally convulsions and death; and a large number of pathologic observations, in which most remarkable elevations and variations of temperature accompany profound alterations and disturbances of the nervous system, without corresponding changes in the respiration and circulation, sustain the theory of centers of control which have their seat in the brain, and which regulate the activity of the spinal cord, (when they are destroyed, the activity of the spinal cord being morbidly increased, as manifested by increased reflex action, quickened respiration, acceleration of the cardiac systole, and increased animal heat), and that a great part of the pathologic phenomena of warmth may be only the expression of the action of the vasomotor nerves, and still farther that the integrity of certain parts of the central nervous apparatus is more necessary for the regulation of animal heat than that of any other part of the body; on the other hand, Breuer and Chrobok, after an investigation of the question, whether the nerves of a part supply the stimulus which causes the febrile elevation of temperature in an inflamed part, by means of experiments on animals in whom they have as far as possible divided all the nerves of one part of the body, think themselves justified in concluding that the fever of the traumatic inflammation is independent of the nervous connections of the inflamed part, with the nerve centers; and by an extended series of observations, (see note) I have established the fact that in hospital gangrene and pyemia, the elevations of temperature are independent of the local lesions of the structures, and are coincident with the introduction of the gangrenous and pyemic poisons into the blood, and the changes of temperature are intimately associated with the changes of the blood and urine, and are invariably accompanied by increased amounts of such constituents as urea, phosphoric and sulphuric acids, which result from the chemic changes of the solids and fluids of the body; and even the traumatic fever accompanying gunshot wounds arises from the intro-

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NOTE.—Investigations upon the Nature, Causes and Treatment of Hospital Gangrene, as it prevailed in the Confederate Armies, 1861-1865, by Joseph Jones. M.D. Surgical Memoirs United States Sanitary Commission, vol. ii, pp. 146-570.

duction of inflammatory products into the blood, and depends not so much upon the extent and nature of the wounds as upon the state of the solids and fluids at the time of the reception of the injury, and upon the character of the inflammatory products absorbed into the circulatory fluid. Even in such inflammatory diseases as pneumonia, attended with extensive tissue change, the introduction of certain organic products into the blood must be active to a greater or less extent in the production of high temperatures.

It is well known that some of the most violent poisons, during their direct action upon the nervous system, are unattended with elevations of temperature; while on the other hand putrid matter, pus and certain animal secretions, as the poison of the rattlesnake and copperhead of this country, induce profound alterations in the constitution of the blood, attended with the most marked variations of temperature; and in the action of the latter class of poisons, the phenomena appear to be akin to the changes which may be induced by ferments, which are not only capable of increasing the amount of heat, but may be limited in their action, or in the amount and character of the changes which they induce in virtue of their chemic constitution and that of the blood or medium in which they are active.

It is well established that certain substances as woorara, coffee, musk and camphor, and putrid fluids, have a direct effect in raising the temperature after their entrance into the circulation.

Voisin and Lionville, by means of subcutaneous injections of woorara, induced a complete artificial fever in human beings, with rigors, heats and sweatings, the temperature rising to 104.8 F., accompanied with all the signs of febrile circulation and secretion and disturbance of the nervous system.

The effects of certain animal substances in elevating the temperature, when injected into the blood, have been shown by the experiments of Billroth, Hufschmidt, O. Weber, Fresse and others. When the putrid solutions or pus are injected in the blood, the temperature rises considerably even within two hours after injection, and reaches its maximum in from two to twenty-eight hours; after a single injection a rapid defervescence generally sets in, shortly after the acme has been reached; while on the other hand, after repeated injections, death constantly occurred generally with high temperatures. Fluids from inflamed tissues, pyemic and septicemic blood, and even the blood of an animal merely suffering from simple inflammatory fever, or from other kinds of fever, produced similar deviations of temperature.

Fresse has determined by experiments, that the rise of temperature induced by the introduction into the blood of the products of decomposition, and of inflammatory tissue destruction, do not depend upon the pus corpuscles, nor upon the so-called germinal matter, which according to some, are such powerful and essential agents in the production of inflammatory diseases, but upon the serum of the fluid; even boiling and subsequent filtration does not destroy the property; and that the removal of the fibrin and even the filtration of the blood of animals suffering from fever, do not deprive it of the pyrogenic effects when injected into the living circulating blood.

The unknown cause which excites the specific morbid process of yellow fever would appear to have something in common with the action of such putrid animal substances, when introduced into the blood; and the fact is worthy of careful consideration that the property possessed by such matters, of exciting chemic change and elevation of temperature, was not destroyed by boiling and subsequent filtration of the putrid matters, for we have thus a clear demonstration that the animalculæ, vegetable or germinal theories, as to the origin of yellow fever, are not *absolutely necessary* to the explanation of the febrile phenomena.

Neither the rapid rise nor the sudden declension of the temperature in yellow fever, therefore, are *necessarily* referable *solely* to the effects of the poison upon the nervous system; because, in the first place, the changes of the blood are among the first manifestations of diseased action, and the progress and termination of each case is largely dependent upon the extent and character of the changes of the blood and the degree of elevation of the temperature; in the second place, the sudden fall of the temperature during the succeeding stage of calm may be referred to the peculiarity of the self-limited chemic changes excited by the poison, and to the structural alterations induced in the muscular tissue of the heart and in the liver and kidneys, and the sedative action of the bile, urea and other excrementitious products retained in the blood upon the nervous system; and finally, in the third place, the changes of the blood and of the heart, liver and kidneys are of a definite physical and chemical nature and could never be induced by a mere exaltation or depression of nervous action, and must be referred to the introduction and action of some agent or material, related in a definite manner in its chemical constitution and physical properties, to the fluids and solids in which it induces these profound physical and chemical changes.

Without doubt, the action of the yellow fever poison upon the nervous system may be indirect and of the most important character; but the best established facts do not justify us in locating the origin of the disease wholly in the action of the poison upon the nervous system, and in fact the earliest sensible manifestations of disordered nervous action, as evidenced in unconsciousness, loss of appetite and chilly sensations may be entirely secondary to the changes in the blood, by which all parts of the nervous system are surrounded and supplied.

It must, however, be admitted that in the present state of our knowledge it is impossible to refer the elevations and depressions of temperature in yellow fever, with certainty to either the decrease or increase of any one constituent of the blood; for M. Andral has shown that when the blood contains more than .004 of fibrin the temperature rises, and in a corresponding ratio. Thus of all diseases, pneumonia is marked with the greatest increase of fibrin, and is the highest in temperature of all the phlegmasiæ; in eighty-five cases, in only thirteen was the temperature below 39 degrees C. (102.2 degrees F.), in forty-four it was between 39 and 40 degrees C. (102.2 and 104 degrees F.) in twenty-six below 40 degrees C. (104 degrees F.) and 41 degrees C. (105.8 degrees F.); and in two rose to 41.2 degrees C. (106.16 degrees F.). In acute pleurisy, in which there is always less fibrin, the temperature only once reached 41 degrees C. (105.8 F.) and usually oscillated between 38.5 degrees C. (101.3 F.) and 39.5 degrees C. (103.1 degrees F). M. Andral, however, records exceptions to this relationship of the increase of fibrin and the elevation of temperature in inflammatory diseases, as in erysipelas, when there have been only .007 of fibrin, the temperature has been 41.8 degrees C. (107.24 degrees F.); and he very justly does not consider the increase of fibrin and the rise of temperature as cause and effect; for in the pyrexiæ, when there is no excess of fibrin, but rather a diminution of this constituent, the temperature is as high or higher than in the phlegmasiæ; thus 42.4 C. degrees (108.32 degrees F.) have been reached in typhoid fever; 42 degrees C. (107.6 degrees F.) in the event of smallpox, the last stage of ague and in glanders, etc. Indeed, the highest degrees are reached in diseases where there is the least fibrin in the blood. (See note.) Neither do the number of red globules affect the rise of temperature to an appreciable degree in inflammations and fevers, as I have carefully determined by comparison of the constitution of the blood, with the elevations of temperature in various diseases.

CHAPTER VI.

CONSTITUTION AND CHANGES OF THE BLOOD IN YELLOW FEVER.

Recapitulation.—Facts illustrating the changes of the blood during the active stages of yellow fever. Alterations of the chemical and physical properties of the albumen and fibrin. In many cases complete disappearance of the fibrin. The disappearance of the fibrin appears to be due, not so much to the action of ammonia, which is so often found in the blood of yellow fever, as to the direct action upon this element, of the febrile poison. Character of the blood in the cavities of the heart after death. The alterations in the physical and chemical properties of the fibrin and albumen, lead to transudations through the excretory structures of the kidneys. While the colored blood corpuscles are not specially diminished in yellow fever, they present under the microscope certain peculiar appearances which are referable to the action of certain extraneous excretory matters in the blood. Extractive and fatty matters increased in yellow fever blood. Bile accumulates in the blood, in consequence of the profound lesions of the liver induced by the febrile poison, and in consequence of the failure in the excretory power of the kidney. The serum presents a golden yellow color in yellow fever. Accumulation of the urinary constituents, and especially of the urea and phosphoric acid, sulphuric acid, chlorid of sodium and carbonate of ammonia, in consequence of the profound lesions induced by the febrile poison and its products upon the kidney. Breath alkalin from presence of ammonia. Blood corpuscles rapidly dissolved, after the abstraction of the blood. Blood of yellow fever undergoes rapid putrefaction. Cases illustrating the changes of the blood in yellow fever. Observations illustrating the changes of the organs in yellow fever and in malarial paroxysmal fever. The colored blood corpuscles are not specially destroyed in yellow fever, as in malarial fever. The nervous symptoms of yellow fever referable chiefly to the presence of bile and the constituents of the blood in the urine. Causes of death in yellow fever. Microscopic character of black vomit. Relations of the variations of temperature and circulation to the changes of the blood. Observations and cases illustrating the relations of the pulse, respiration and temperature. Observations of various observers, as Rush, Devoe, Currie, Caldwell, Samuel Jackson, Lewis, Arnold, Chalmers, Lining, Daniel Blair, John Davy, Faget, Leighton and Lyons. The cause of the rapid rise and declension of the temperature in yellow fever must be sought chiefly in the changes induced in the blood, and in the organs upon which the circulation and integrity of the blood depends. Neither the rapid rise, nor the sudden declension of the temperature can be referred wholly to the effects of the yellow fever poison upon the nervous system. Discussion of these propositions.

That the blood undergoes profound changes during the period of febrile excitement of yellow fever is manifest even to the casual observer, in the impeded capillary circulation, purplish, jaundiced and dusky hue of the surface, livid blotches, passive hemorrhage from slight abrasions, leech bites, blistered surfaces, and hemorrhages from the ears, eyes, mouth, gums, and gastro-intestinal mucous membrane, which in some cases are characteristic of the succeeding period of calm or exhaustion.

Although after the subsidence of the fever, at the end of from two to five days, the skin becomes cool and pleasant, the tongue shows a disposition to clean, and the tips and edges are less red, the thirst abates, and appetite for food returns, and the anxiety and morbid fear of death, which may have been great, subside, and both patient and bystander may regard convalescence as established; nevertheless, a careful examination, will show that the circulating fluid has been altered during the preceding stage of febrile excitement; the eye loses its glistening appearance and assumes a condition of chronic vascularity, of a dull orange red; the flushed countenance gives way to a bloated appearance and dusky, dirty complexion; the sclerotic of the eye is jaundiced, the forehead presents a dusky appearance, which extends also over the neck and chest, the languor of the capillary circulation is indicated in the purple and lobster red condition of the skin, and by the pale marks left by pressure over the forehead, cheek, abdomen, or surface of the extremities; the matters vomited, which at first may be tinged with bile, change to a clear, acid, mucous fluid, and become discolored by small dark specks and flocculi of blood. The further changes of the blood are indicated by an increase in the purplish or yellow suffusion of the surface, and by such a loss of vitality and of the fibrinous elements as manifests itself in the raw claret-colored surface of blisters, in epistaxis, ecchymosis, bloody oozings from the mouth, ears or anus, excoriation of the scrotum, the copious ejection of dark altered blood from the stomach, with little or no apparent effort, copious stools of black altered blood, and by the foul fetid alkalin breath containing ammonia.

It appears to be an error to treat of the changes of the blood as confined to the latter stages of yellow fever, thus regarding the disease as manifesting only two grand stages, viz: that of reaction, irritation and fever, and that of unhealthy subsidence or contamination, characterized most prominently by exhaustion of the nervous system, slow pulse and passive hemorrhages.

The changes of the blood appear to be continuous, from the time of the introduction of the poison to the fatal termination; these changes being increased, and their character being modified as the disease advances, not only by the direct action upon the constituents of the blood by the poison, but also by the addition of certain noxious substances, as bile, urea, carbonate of ammonia, sulphates and phosphates and extractive matters.

Certain constituents of the blood, as the albumen and fibrin, are not only altered physically and chemically in the early stages of yellow fever, but as the disease advances, from the cause just specified, certain excrementitious matters, which in a state of health are continually eliminated accumulate in the circulating fluid, and by their direct action upon the elements of the blood, and upon the nervous system, and by their disturbing action upon the processes of digestion and nutrition still further alter the physical, chemical and vital properties of this fluid.

As far as my observations extend, the alterations of the blood in yellow fever consist chiefly in:

1. Such an alteration of the chemical and physical properties of the fibrin and albumen as leads to the transudation of the latter through the excreting structures of the kidney.

2. Various degrees of alteration and diminution of the fibrinous element.

In some cases there is an almost entire disappearance of the fibrinous element. This disappearance of the fibrinous element appears to be due not so much to the action of ammonia, which is so often present in abnormal amount in the blood of yellow fever, but in the direct action upon this element of the febrile poison. From this alteration in the amount and character of the fibrinous element it results that the blood coagulates imperfectly in most cases, and the clot is voluminous and soft. The amount of serum formed is small, and upon standing, the clot frequently dissolves, leaving a thick, non-coagulable grumous blood. The blood taken from the cavities of the heart and large blood vessels after death is frequently black and fluid; and if, as is sometimes the case, fibrinous concretions are formed in the cavities of the heart, they are small, soft, and of a bright golden yellow color, and much smaller in size and less firm than is usual in attendant diseases near the fatal issue, with similar retardation of the circulation in malarial fever. In the latter disease the formation of firm, light colored blood clots is, as I have shown, by a large number of observations, not only frequent, but also a cause of death in certain cases of pernicious malarial fever.

3. While the colored blood corpuscles are very slightly diminished in yellow fever, they present under the microscope, certain peculiar appearances, which appear to be referable to the action of certain extraneous matters in the blood.

4. Increase of the extractive matters of the blood.

5. Increase of the fatty matters.

6. Accumulation of bile in the blood, in consequence of the profound lesions of the liver induced by the

febrile poison, and in consequence of the failure of the excretory function of the kidneys. Many of the changes of the blood, as well as certain cerebral symptoms, may be dependent upon the presence and action of the biliary constituents. Even the nausea and vomiting, as well as the depression of the pulse, and the nervous agitation, delirium and coma, may to a certain extent be referred to the same cause. The serum presents a golden color in yellow fever. This condition of the serum is due to the presence of bile and may be present also in grave cases of paroxysmal malarial fever. If a drop of yellow fever blood be allowed to fall on a piece of bibulous paper the center will appear of a brilliant scarlet, while around the central accumulation of colored blood corpuscles extends a ring of bright golden colored serum.

7. Accumulation of the urinary constituents, and especially of the urea and phosphoric acid, sulphuric acid, chlorid of sodium and carbonate of ammonia, in the blood, consequent upon the profound lesions induced by the febrile poison and its products upon the kidneys. Not only is the blood alkalin in many cases of yellow fever, but the blood contains ammonia, resulting from the decomposition of the urea, the presence of which may be rendered evident by various means, as by the addition of potassa, soda or lime; and in some cases the ammonia is so abundant as to be demonstrable without resorting to these reagents. As the phosphoric and sulphuric acids are retained in the blood when the function of the kidneys is embarrassed or suppressed, they unite with the ammonia and thus diminish the alkalinity of the blood and render the addition of potassa or soda or lime necessary in certain cases for the demonstration of the ammonia. The reaction of the yellow fever blood is alkalin during life, but it rapidly changes in some cases to the acid reaction after death, from the rapidity of the putrefaction, and also from

the development of numerous forms of low organization.

8. Rapid dissolution of the colored corpuscles, after the blood is abstracted from the body, either during life or after death. The rapid alteration of the investing membrane of the colored blood corpuscles in the blood of yellow fever, after the abstraction of the blood from the vessels, appears to be intimately related to, if not absolutely dependent upon, the physical and chemical action of the biliary and urinary constituents retained in the blood. During life the blood corpuscles, in virtue perhaps, of their vital endowments and of their relations to the oxygen received during respiration, resist the solvent action of the bile, urea and ammonia; but after the blood is abstracted and loses its vitality and is exposed in vessels these agents excite their characteristic actions. It results also that in many cases the serum separating from the clot presents a bloody florid color, not only from the incomplete separation of the colored corpuscles during the process of coagulation, but also from the dissolution of the globules, and the escape of the coloring matters of the blood. I have recently embraced the opportunity of testing the effects of human bile upon the blood corpuscles. I selected for this inquiry the blood of the *amphiuma* in which animal the globules are comparatively of great size. The bile rapidly dissolved the investing outer membrane, and liberated the internal nodulated nuclei.

9. Rapid putrefaction of the blood of yellow fever, after its abstraction from the living body, or from the large vessels after death.

These conclusions have been established by careful and laborious observations at the bedside and in the laboratory; the nature and method of which will be illustrated by the following observations:

The following analysis of the blood in a severe case of yellow fever which terminated fatally, on the sixth day, with black vomit and urinary suppression, presents a correct view of the blood composition on the fourth and fifth days, in severe cases of this disease.

The fibrin in 1,000 parts of blood extracted from the arm of this patient being 0.271, or not more than one-tenth of that of healthy blood. This diminution of the fibrinous element was attended by passive hemorrhages, black vomit, etc.

Specific gravity of blood, 1055.6; of serum,	1027
Water in 1,000 parts of blood	802.12
“ “ “ “ serum.	922.90
Solid matter in 1,000 parts of blood	197.88
“ “ “ “ “ serum.	77.10
“ “ “ serum of 1,000 parts of blood	67.03
Saline matters in 1,000 parts of blood	8.48
“ “ “ blood corpuscles of 1,000 parts of blood	1.78
“ “ “ 1,000 parts of serum	7.71
“ “ “ serum of 1,000 parts of blood	6.69

1,000 parts of blood contained:

Water.					802.12
Solid residue, 197.88	Organic matters, 189.40	Dried blood corpuscles, 130.57	Organic matters		128.79
			Saline matters.		1.78
	Fixed saline constituents. 8.48	Solid matters in serum 67.03	Albumen . 53.40	Organic matters	51.59
				Saline matters .	1.81
			Extractive matters 13.72	Urea. bile, fat and carbonate of ammonia, etc.	8.84
				Inorganic matt'rs	4.88
	Fibrin.				0.271

1,000 parts of blood contains:

Moist blood corpuscles, 522.28	Water of moist blood corpuscles		392.71
	Organic matter.		128.79
	Saline matter		1.78
Liquor sanguini. 477.72	Albumen. . . 53.40	Organic matters.	51.59
		Saline matters.	1.81
	Extractive matters . . 13.72	Urea, bile, fat and carbonate of ammonia	8.84
		Inorganic matters	4.88
	Fibrin		0.271

Urea and bile acids were detected after death in considerable amounts in the brain, liver, heart and spleen of this patient; the black vomit also, ejected during life and remaining in the stomach after death contained urea.

John Alter, yellow fever. Patient attacked with symptoms of yellow fever, September 29, died Oct.

4, 1871. Analysis of blood, black vomit and bile.. Urea detected in the blood during life, and also after death. Urea detected in large quantities in the brain, in the fibers of the heart, and in the liver and spleen. Fat, in the form of globules, deposited in large amount in the liver, kidneys and in the muscular structures of the heart. Tubuli uriniferi filled with granular fibroid matter, oil globules, and epithelial cells. Blood and brain and all the organs contained bile. Stomach congested ; mucous membrane softened, epithelial cells filled with granular matter.

Case 1.—John Alter, age 21; native of Switzerland; has resided in Louisiana during the past four years, and has lived alternately on the shores of Lake Pontchartrain, and in New Orleans; the greater portion of his time being spent at the Lake. Entered the Charity Hospital Oct. 3, 1871, 10 o'clock A.M. Patient lethargic and dull as if suffering under the effects of some narcotic poison ; in response to careful and persistent inquiries, however, he gave with difficulty the following facts relating to his disease : "Took passage on steamboat to Vicksburg ten days ago for the purpose of obtaining employment; failing to effect his object, at the end of two days, started back on a steamboat, and four days âgo, while on the return trip, began feeling unwell." Patient complains of pain over region of the stomach, in the small of the back and in the lower extremities. Has a sleepy heavy look ; complexion yellow and dusky ; conjunctiva of eyes, mucous membrane of lips and gums, and the skin generally, greatly congested, tongue furred in the center and on the borders ; bowels constipated. Beef-tea, small quantities of alcoholic stimulants and absolute rest were ordered. Patient appeared to be quiet during the day, but was very restless during the night. October 4, 9 A.M., patient delirious, restless ; ejecting black vomit from the mouth, with little apparent effort. Complexion bright yellow ; whites of eyes yellow ; skin greatly congested, and in the extremities mottled ; eyes congested ; black vomit and black blood from the gums trickling down the corners of the mouth. Body emits a most disagreeable odor, resembling that emitted in some of the worst cases of typhus fever, smallpox and typhoid fever. Pulse full and slow, skin only moderately warm, dry and harsh to the feeling. When pressed with the finger, the blood is driven out of the capillaries, but slowly returns into the discolored spot. Complete suppression of the urinary secretion during the past twenty-four hours.

I determined to bleed this patient, and to make a careful microscopic examination and chemic analysis of the blood.

When the arm was tied up preliminary to the use of the lancet, the patient cried out and gave forth delirious screams mingled with incoherent sentences and curses, and at the same time struggled violently. After the vein was opened, it required all the strength that I could command, with one hand, to extend the right arm and keep it in the proper position for the collection of the blood in suitable vessels.

MICROSCOPIC AND CHEMIC EXAMINATION OF BLOOD.

The color of the venous blood was purplish, between that of arterial and venous blood. When a drop of blood was allowed to fall upon a sheet of white bibulous paper, a central bright red spot remained with a surrounding bright golden aureole of serum, which spread around the central corpuscles by capillary action. The blood coagulated very slowly and formed a large loose coagulum, or clot, which contracted slowly and imperfectly. Thus in the 1,000 grain specific gravity bottle the coagulum filled the whole bottle, and from this amount of blood not more than 150 grains of golden colored serum could be collected at the end of forty-eight hours. The blood corpuscles tended to rapid dissolution in the serum, and upon long standing the serum changed from this cause to a bright red. The reaction of the blood was most carefully determined as it flowed from the veins, and was found to be slightly alkalin, as is usual in healthy blood. I regarded this observation with interest as in several cases, in which I had abstracted blood from the cavities of the heart, after death, it gave a decided acid reaction; but the present observation would seem to show, that the acid reaction was due to post-mortem changes. Immediately after its abstraction, the blood was subjected to a careful and rigid microscopic examination. Under a magnifying power of one-fifth of an inch (Smith & Bucks, of London, best objective glass). many of the blood corpuscles presented an irregular stellate outline. When viewed under high magnifying powers, as the one-eighteenth inch immersion lens of G. & S. Merz, of Germany, with eye glasses, to magnify 1,050 diameters, the crenated and stellate blood corpuscles were found to be studded upon the surface, with nodular rounded projections. The colored blood corpuscles appeared to be undergoing changes of form, as if nodular transudations of the globulin were forming upon the surface. These changes were most marked and frequent, upon the surface and outer portions of the clots, and resembled in some respects the amœboid movements of the coalescing corpuscles; the nodules were, however, uniformly diffused over the surface of the corpuscles. When the blood was examined, from the interior of the clot, the corpuscles were found conglomerated together forming rolls or piles, adhering together by their flat surfaces, like the rouleaux of the blood of inflammatory diseases and of the horse. The corpuscles which had been joined and agglutinated together by their flat surfaces, were normal in shape, and presented no stellate or nodulated outline as was the case with the corpuscles from the surface of the clot, and from the surrounding golden serum. It appeared as if the nodulated exudation had formed the bond of cement between the opposing flat surfaces. Upon standing twenty-four hours and longer, the colored corpuscles tended to dissolve and lose their outline, and the serum became colored from the escape of the coloring matter of the red globules.

The colored corpuscles appeared to be acted upon and altered by the urea and bile which chemic analysis revealed in considerable amount in the serum. After standing in an open beaker or in porcelain capsules for forty-eight hours, numerous vibrios made their appearance, as in other putrefying animal fluids, as blood, albuminous urine, and serous exudations. But no living animalculæ or vegetable cells or sporules or pigment granules were discovered even after the most diligent search with high powers, ranging up to the twenty-thousandth of an inch objective, in the fresh blood. The blood drawn from the arm during life putrefied much more slowly than that taken from the heart and large blood vessels after death. The blood was carefully examined by the best and most reliable chemic methods, and was found to contain a comparatively large amount of this constituent; large and well formed crystals of urea, and of the nitrate of urea, were obtained from comparatively small quantities of blood.

As much urea was obtained from 100 grains of the yellow fever blood as is ordinarily obtained from 7,000 grains of healthy blood. The serum was also carefully tested for bile, and was found to contain all the ingredients of bile. The blood also contained numerous small oil globules and in one of the specimens a distinct oily scum rested upon the surface of the clot. So striking was the oily appearance of the blood that it was noticed by the medical students who assisted during the bleeding. The presence of the urea in increased amount, and of the bile in the blood, were due to the suppression or arrest of the functions of the kidneys and liver. To the presence of these substances in the blood must be attributed in part, at least, the delirium, intoxication and aberrated muscular and nervous action, and to a certain extent the nausea, vomiting, gastro-intestinal irritation and black vomit. The altered shape of many of the colored blood corpuscles was certainly due, in a great measure, to the physical and chemical action of the urea and bile of the serum. Specific gravity of venous blood from the arm of this yellow fever patient, 1055.6. The density of this blood was somewhat below that assigned by physiologists and pathologists as the standard of healthy blood. Thus Becquerel and Rodier give the density of the defibrinated blood of the male as 1060, and of the female as 1057.5.

The specific gravity of the serum of the venous blood from the arm of this yellow fever patient was 1027. The specific gravity of the serum, therefore, did not differ materially from that of health; according to Becquerel and Rodier, the density of the serum of the male being 1028, and of the female 1027.4.

The serum was of a deep yellow color, similar to the yellow color of the liver and of the diluted bile from the gall bladder of yellow fever subjects. Clot, soft, large, and without any very great contractile power. After the contraction of the clot the serum was tinged of a red color, from the presence of colored corpuscles and the coloring matters of the blood. The amount of serum forced out of the clot was relatively small and appeared to possess the power of dissolving the colored corpuscles upon standing in contact with the clot, the red color increasing gradually in depth. In these respects the yellow fever blood presented a marked contrast to healthy and inflammatory blood. Only 108 grains of serum could be obtained from a vessel containing 890 grains of blood.

Fibrin in 1,000 parts of venous blood abstracted from the arm of this yellow fever patient, 0.271. The fibrin was greatly diminished, being only one-tenth of the quantity present in the normal blood. The fibrin itself appeared to be of the usual tenacity, and the imperfect contraction of the clot appeared to depend rather upon the small amount of fibrin, than upon any physical or chemical change in the constituents of the blood. In the marked diminution of fibrin, we have a most interesting and important explanation of the cause of the hemorrhagic tendency in yellow fever.

The following are the chief characteristics of the blood in the stage of depression in yellow fever, as established by the preceding chemic analysis:

Specific gravity of blood and serum not specially altered.

Blood coagulated slowly and imperfectly.

Clot voluminous and soft.

Fibrin, quantity deficient and not more than one-tenth the normal amount.

Reaction of blood alkalin.

No deficiency of colored blood corpuscles, the dried blood corpuscles being 130.57, and the moist blood corpuscles 522.28. The organic matters of the moist blood corpuscles are normal in amount, while the fixed saline constituents are diminished, being only 1.78 in the blood corpuscles of 1,000 parts of blood.

The relation of the moist blood corpuscles to the liquor sanguinis, as far as quantity was concerned, was not disturbed, the former standing in relation to the latter in the 1,000 parts, as 522.28 of the blood corpuscles to 477.72 liquor sanguinis.

The albumen of the liquor sanguinis was diminished to a marked degree, being only 53.40 parts in 1,000 parts of blood. The extractive matters of the liquor sanguinis were increased. The extractive matters were complex, and included bile, urea and ammonia. The fixed saline constituents of the liquor sanguinis was not diminished. Without doubt the loss of albumen, as well as the presence of bile, urea and other excrementitious matters in the serum or liquor sanguinis, determine to a certain extent, at least, the character of the capillary circulation and the production of black vomit. The patient died twelve hours after this observation. The kidneys never acted and he appeared to die from the combined action of the febrile poison, bile, urea and other excrementitious matters, and the loss of blood during the slow but persistent hemorrhage from the gastro-intestinal mucous membrane.

Autopsy twelve hours after death: Exterior, superior portions of face, trunk and upper portions of limbs of a deep golden orange color. The lower dependent portions of the face and of the trunk and limbs were of a mottled, purplish, yellow and livid maculated appearance, as if the patient had been beaten with a club. The general appearance of the exterior of the body was horrible. Black vomit had run out of the corners of the mouth, and saturated the winding sheet, and had trickled in a muddy filthy stream along the sides of the face and neck. Features full and somewhat swollen. Belly full and tympanitic. Well formed muscular man, in the full vigor and strength of manhood. Large well developed chest, with full pectoral muscles. Arms full and round, with good muscular development. The left knee appeared to have been injured at some previous time, at least months if not years before the fatal illness. The patella was displaced downward and outward laterally, and the joint was ankylosed. A small ulcer existed upon the outer aspect of the left knee joint, below the edge of the displaced patella. The left leg was a little shorter and smaller than the right. I had the bones carefully cleaned after death, and found that they had been altered by chronic inflammation, which had been attended with certain changes of the bony structure. This injury was of long standing, and had nothing whatever to do with the present attack, as the patient was able to perform active labor up to the time of his last illness, and the parts in and around the joint presented no swelling or discoloration. The patient appeared to have been suddenly cut off in full vigor and health, and in death resembled one into whose veins a deadly poison had been injected by a venomous reptile, which had not only poisoned, but also enveloped in its powerful coils and strangled its victim.

Head: Dura mater, pia mater and arachnoid membranes congested with blood. The brain was congested with blood, not only in its membranes but also throughout its textures. The brain and its membranes, however, presented no marks of inflammation —no exudation. Membranes of spinal cord congested with blood. The congestion of the blood vessels of the brain appeared to be similar in all respects to that of the capillaries and small blood vessels in the different organs and tissues. The consistency of the brain was normal. Weight of brain, two pounds and fourteen ounces; it was carefully tested for bile and urea. The infusion of the structures of the brain presented a bright golden color, like that of the serum. When carefully tested, the presence of the coloring matters and acids of the bile was conclusively determined in the cerebral structures. When the decoction of the brain was evaporated in a watch-glass and examined under the microscope, numerous beautiful stellate and prismatic crystals of the phosphates of lime, and ammonia and magnesia made their appearance.

When carefully tested for urea, according to the best methods known to chemists, the alcoholic extract obtained from the water extract was found to be literally loaded with the product of the metamorphosis of the tissues, and as large a crop of perfectly formed and characteristic crystals of the nitrate of urea were obtained as from an ordinary specimen of urine of moderate specific gravity. The brain contained more urea than any other organ, the liver not excepted. It was clearly established by this chemic examination that the urea existed in much larger quantity than in the healthy brain, or in the brains of those who die from diseases in which the action of the kidneys is not diminished.

Thorax: The pericardium was congested. As is usual in yellow fever, the capillaries and small blood vessels of the pericardium presented a beautifully arborescent appearance from the accumulation of colored corpuscles. I have in my possession a portion of the pericardium of a yellow fever patient, which I preserved by simply spreading upon a glass slide, and after drying, saturating with Canada balsam and covering with a glass slide. The preparation shows an intense congestion of the blood vessels and capillaries, and so numerous are the colored corpuscles, and so brilliant the colors, that the preparation resembles the best injections with the most brilliant carmine. The pericardial sac contained about two ounces of bright golden colored transparent serum.

The heart presented a brownish-yellow appearance, resembling the pale flabby heart of scurvy. The textures although softer than in the normal heart, were, however, firmer than in well marked fatty degeneration. The muscular textures of the heart required considerable pounding in the mortar to reduce them to fragments sufficiently small for decoction. The decoction of the heart in distilled water was loaded with golden-colored oil, which floated in a thick scum upon the surface, and it resembled in its rich oily nature the richest soup made from the marrow and fat of the bones of fat well fed animals. The fat was far more abundant than is usual in the heart of malarial fever or other diseases in which there is no fatty degeneration of this organ. The filtered decoction of the heart was of a golden yellow color. The decoction of the heart contained bile in abundance. As the decoction of the heart, after careful filtration, slowly evaporated in a porcelain capsule, a deep orange-colored ring formed around the sides of the vessel. Weight of heart, eight and one-half ounces. Both cavities of the heart contained blood. Several small, ropy, golden-yellow clots were also discovered in the cavities of the heart. The muscular tissue of the heart appeared to have lost its tenacity and to have become relaxed and somewhat softened; and this view was confirmed by microscopic examination. The heart was also carefully tested for urea, and this substance was clearly detected in considerable quantity, but in relatively less amount than in the brain. Under the microscope, oil was found in the form of minute globules, diffused through all the tissues of the heart, and even within the ultimate muscular fibers. The muscular fibers of the heart presented under the microscope, a granular appearance, and the transverse striæ were not as distinct as in normal hearts. The increase of free fat or oil in the structures of the heart was therefore determined by the microscope and by actual experiment. The heart contained therefore in comparatively large amounts, bile, urea, oil.

No animalculæ or vegetable cells or spores were observed under the one-eighteenth inch immersion objective of Merz, either in the blood or textures of the heart.

Lungs: The lungs in the dependent portions were greatly congested with blood, and presented a mottled purplish and red appearance. The lower portions of the lungs, when cut, resembled the congested lungs of pneumonia, and in circumscribed portions effusions of blood had taken place.

The muscular structures of the thorax, when cut and exposed to the atmosphere, presented a brilliant scarlet hue, like those of patients poisoned with cyanid of potassium.

Abdominal cavity: Alimentary canal, exterior surface of stomach somewhat congested. Internal mucous membrane of a deep purplish and brownish-purple and red color, with variations in the depth of the color, presenting a mottled appearance. Under low magnifying powers, suitable for the examination of the mucous membrane of the stomach by direct light, an intense congestion of the capillaries and small blood vessels was observed.

In some places the epithelium of the mucous membrane of the stomach had been removed, and the capillaries were exposed and ruptured, and from the points of rupture issued small flakes or coagula of blood, resembling those so frequently found in black vomit. The epithelial cells of the mucous membrane were detached in large numbers; they could also be removed readily by slight and delicate scraping with a spatula.

Black vomit: The stomach contained one pint of black vomit, of a dark purplish color, resembling in all respects defibrinated and altered blood. Under the microscope, the black vomit was found to contain numerous colored blood corpuscles, variously altered, also cells from the mucous membrane of the stomach, and also broken capillaries. In this case the black vomit resembled in all respects blood which had been defibrinated and mixed with gastric juice, or with a mucoid fluid. This specimen of black vomit, taken from the stomach twelve hours after death, contained some fibers, and the sporules and thallus of a plant resembling the yeast plant. The yeast plant appears to be most abundant in the acid black vomit of life, and appears to be dependent in large measure for its rapid development upon the articles of food consumed. If much sugar be introduced into the stomach with the food, the growth of this plant is rapid in a stomach which has lost its muscular and digestive power to a great extent. The sporules of these plants appear to be ever present in the atmosphere, and they increase rapidly whenever the proper conditions, as in the disabled stomach of yellow fever with its albuminous contents, exist for their growth and development. The odor of this specimen of black vomit was putrid and disgusting, resembling that of decomposing blood. When heated upon the sand-bath the odor emitted was very disgusting and overpowering, and excited nausea. Specific gravity of black vomit 1026. Reaction of black vomit alkalin. The surface of the mucous membrane of the stomach, as well as the black vomit itself, emitted strong fumes of ammonia, in the form of free ammonia, and its carbonate and sulphid. When a glass rod was dipped in strong hydrochloric acid and held over the mucous membrane of the stomach and over the black vomit, heavy dense fumes of chlorid of ammonium were evolved. The alkalin character of the black vomit was evidently due to the decomposition of the urea, and its elimination as carbonate of ammonia by the gastric mucous membrane. The black vomit was carefully tested for urea, and yielded this substance in considerable amount, as well as ammonia and its carbonate and sulphid.

Analysis of black vomit: Reaction alkalin; alkalin reaction dependent upon ammonia; emits heavy fumes of chlorid of ammonium in the presence of hydrochloric acid; specific gravity of black vomit 1026.

One thousand parts of black vomit contain:

Water.				906.00
Solid residue, 94.00	Organic matters, 85.40	Albumin, colored blood corpuscles, mucous corpuscles and epithelial cells, 77.14	Organic matters	75.46
			Saline matters	1.68
	Saline matters, 8.60	Extractive matters, urea, ammonia, etc., 16.86.	Urea, ammonia and fatty matters	9 94
			Saline matters	6.92

Still further to confirm the existence of blood in the black vomit, the ash derived from careful incineration was tested for iron, and was found to contain this substance in amount equal to that which would be found in 77 grains of blood.

Microscopic and chemic examination of blood from cavities of the heart: The blood from the cavities of the heart contained several loose, golden-colored fibrinous concretions. The blood was fluid and did not coagulate upon standing. The odor of the blood twelve hours after death was putrid, but not so disagreeable as the black vomit taken from the stomach after death. The colored blood corpuscles were crenated and altered in form in some instances; many of the corpuscles, however presented the usual appearance, while others were more round and smaller than in health. The blood from the cavities of the heart and from the large blood vessels contained numerous fibers. In twelve hours after death the blood manifested even within the heart itself the presence of living vibrating animalcules. Upon standing, these increased in numbers. They were also, as we have just seen, present in the black vomit of the stomach. The fibers must have been formed or originated in the following ways:

1. They existed in the blood before death, being received through the pulmonary apparatus. We can not adopt this view, because they are entirely absent from the fresh blood examined during the life of the patient.

2. They arose spontaneously in the blood after death, as a result of the putrefactive process. The adoption of this view would involve the adoption of

the theory of spontaneous generation, which, as far as our investigations have extended is, in the present state of our knowledge untenable.

3. They penetrated from the stomach into the blood, finding ample space for entrance through the broken capillaries and through the eroded surface of the gastric mucous membrane.

The germs of the fibers existed in the tissues during life, and even in a state of profound health. They were prevented from germinating by the chemic changes (compositions and decompositions) characteristic of health. After death they multiplied rapidly and found a ready entrance from the tissues into the blood vessel system through the pores of the capillaries and small veins, described by Conheim. These so-called stomata vary in size from 1-1000 to 1-10,000 of an inch in diameter; and if it be true that there are really openings or pores in the small veins, we have an explanation of the mode in which the minute vibrios might readily find entrance into the blood from the surrounding tissues. The surface of the blood from the heart presented an oily appearance, and the microscope showed the presence of minute globules. When a drop of the blood was allowed to fall upon a piece of white filtering paper, the center presented a bright red appearance from the presence of the colored corpuscles, while the central colored spot was surrounded by a bright red golden colored border.

The reaction of the blood from the cavities of the heart was acid.

Chemic analysis of the blood revealed similar results to those obtained during life; thus, the colored blood corpuscles were normal in amount; fibrin greatly diminished, and in fact entirely absent; bile and urea present in large amount. The fact that the fibrin was diminished even in the acid blood would establish the important fact that the diminution of this element in the yellow fever blood was real and not apparent, and dependent merely upon the presence of ammonia. The acid reaction of the blood appeared to have been the result of post-mortem change and may have been induced by the vibrios. The acid reaction may also have depended to a certain extent upon the relation of phosphoric and sulphuric acid and acid phosphates in the blood, in consequence of the arrest of the action of the kidneys. The brilliant scarlet color of the blood upon exposure to the atmosphere, and of the muscles of the thorax without such exposure, may be due to the presence of urea and phosphoric acid and acid phosphates, the accumulation of which in the blood was dependent upon the arrest of the function of the kidneys. To the same cause, that is the cessation of the action of the kidneys, must be referred the remarkable bloodiness of the tissues; the watery element of the blood is not properly removed by these organs and the blood vessels become in consequence, filled and distended with the altered blood. The passive hemorrhages observed in the various organs, stomach, lungs and even in the muscular structures, must be referred to the destruction of the fibrin of the blood and to the great destruction of the blood in virtue of the cessation of the action of the kidneys, liver and stomach. It results also from the same causes, viz.: The fluid character of the blood and the great distention of the blood vessel system with an abnormal amount of altered blood, so that when a canula is plunged into and through the walls of the heart of a yellow fever subject, even hours after death, the blood will flow in a continuous stream through the canula, and some pints may thus be drawn off.

Spleen: Somewhat enlarged and softened. Weight ten ounces. Splenic pulp and structures changed readily and rapidly to the arterial hue when exposed to the oxygen of the atmosphere. Under the microscope, the splenic pulp was found to consist of altered red corpuscles, granular masses and numerous oil globules. The decoction of the spleen presented a brownish, purplish, reddish, mahogany hue, quite different from the golden hue of the decoction of the heart and liver. Upon chemic analysis the spleen contained both bile and urea.

Liver: The liver presented a yellow and brownish-yellow appearance. The portal system of capillaries appeared to be congested with blood. A few small slate-colored spots were observed upon its surface. The structures of the liver were firm and appeared to be firmer than in health; considerable force was required in mashing the structures in the wedgewood mortar previous to analysis. Upon analysis the liver yielded animal starch, grape sugar, urea in considerable amount. The decoction of the liver yielded urea to a less amount than the decoction of the brain. Under the microscope, the liver was found to be loaded with oil globules, and many of the cells were greatly distended with yellow oil. The oil globules were also deposited within the meshes of the fibrous tissue of the liver. Owing to the large amount of oil present within and around the liver cells, they were indistinct in their outlines. The accumulation of oil in the liver was undoubted. The color of the liver, especially upon the cut surface, was that characteristic of yellow fever, and its consistence was as usual, also as is usual in this disease, increased. It would appear that albuminoid or fibroid granular matter transudes through the blood vessels of the liver, as through those of the kidney. It is probable that the peculiar alterations of the liver in yellow fever, cause obstruction to the free flow of blood through its hepatic and portal veins and capillaries, and in such obstruction we may find some explanation of the congested portal circulation, and even of the gastro-intestinal hemorrhage, or at least a favorable condition to its establishment.

Bile: Specific gravity of the bile from the gall bladder, 1040. The bile presented a dirty, greenish-yellow hue, was thick and grumous, and under the microscope was found to contain numerous cells of the mucous membrane of the gall bladder. Mucous membrane of gall bladder deeply congested, and at the same time discolored by bile. When viewed in mass the bile presented a greenish-black color. It was highly concentrated; a few drops being sufficient to color of a golden color half a gallon or more of water. As is usual in yellow fever, tbe amount of bile in the gall bladder was small, only 120 grains being obtained. This diminution of bile appears to be characteristic of yellow fever; the gall bladder as a general rule is small and contracted, and contains a small amount of bile, rarely exceeding 150 grains, and rarely not reaching 90 grains in intermittent, remittent, pernicious and malarial hemorrhagic fever, and in typhoid fever, on the other hand, the bile is more abundant; and as a general rule the gall bladder after death is distended with bile. In yellow fever, bile is also absent from the intestinal canal, and it would appear from these facts, as well as from the jaundice of yellow fever, that in this disease there is an occlusion of the hepatic ducts, in a manner similar to the occlusion of the excretory ducts of the kidneys.

Result of chemic examination of bile: Specific gravity of bile, 1040. The specific gravity of the bile was above that of the serum. Did not coagulate by heat. The proper reagents failed in producing any crystalline constituents.

Solid residue in 120 grains of bile:

The whole amount contained in the contracted gall bladder . . . . grains	15.66
Solid residue in 100 parts of bile	13.05
" " " 1000 " " "	130.50
Water " 1000 " " "	869.50

Kidneys: Weight of kidneys, ten ounces. Kidneys congested on the exterior, more especially in the cortical portion, but of a more decided yellow color than in the kidneys of health. When sections were made the kidneys presented a yellowish color, resembling kidneys undergoing fatty degeneration. The kidneys, with the exception of a few circumscribed, small slate-colored spots upon the exterior, presented an appearance and color similar to that of the heart and liver. Under the microscope, the excretory cells and tubuli uriniferi of the kidney were found to be loaded with oil globules and granular, fibroid and albuminoid matter. The oil globules were diffused throughout the excretory cells, tubuli uriniferi, Malpighian corpuscles and fibrous textures of the kidney. The arrest of the urinary excretion, appeared, after prolonged and careful microscopic examination of sections of the kidney, with Valentin's knife, to have been due to the filling up of the Malpighian corpuscles and tubuli uriniferi and excretory cells with oil globules and granular, albuminoid or fibroid matter; and also to the stagnation and conglomeration of the colored blood corpuscles in the delicate capillaries of the kidney. The capillaries of the kidneys were filled with colored blood corpuscles.

The bladder contained no urine, and it was evident that the patient had not excreted any urine during the last forty-eight hours of life, and many of the symptoms during life were without doubt due to the arrest of the urinary secretion.

We have thus presented the results of the laborious and careful investigation of this case, which required my undivided attention for the entire period in which it was under observation, to the exclusion of all other business or interruption, with the exception of a few hours devoted to sleep and meals, not exceeding seven hours out of the twenty-four, each day, the remaining seventeen being devoted to the investigation; in order that a true picture might be presented of the relations of the changes of the blood to those of the various organs. Each case studied in this manner becomes a living exponent and demonstration of the nature of the disease, just as the description of one animal will to a great extent serve as the accurate description of the entire species to which it belongs.

Case 2.—Charles Collingberg; native of Iowa; attacked with yellow fever July 30, 1871; died in Charity Hospital after the supervention of black vomit August 4. Came to New Orleans four weeks previously; occupation, flatboatman. Supposed to have contracted the fever on the bark *Mary Pratt*, from Cienfuegos, which had arrived a short time before with sugar. Is said to have slept for four nights previous to his attack upon the *Mary Pratt*.

According to official reports, this was the first case of yellow fever which occurred in this city during the summer of 1871. Although the number of cases of yellow fever which occurred in the summer and fall of 1871 was small, yet the disease appeared to be of a violent and fatal nature; thus up to October 6, out of thirty-seven cases, which occurred during the preceding sixty-nine days, sixteen terminated fatally, thus giving a mortality of 43.2 per cent.; and the active and fatal character of the disease was still further manifest in its comparatively short duration; thus of these sixteen fatal cases, one terminated on the third day; one on the fourth; two on the fifth; five on the sixth; four on the seventh; one on the eighth; one on the ninth, and one on the sixteenth day.

According to the official reports of the Board of Health, 114 well marked cases of yellow fever occurred from the date of the first case, July 30, to that of the last, December 18, and of this number fifty-five terminated fatally; that is the mortality reached the extraordinary figure of one death in 2.07 cases, or 48.24 per cent.

In the case now under consideration, (Geo C. Collingberg) the blood from the cavities of the heart, as well as the black vomit, and the structures of the heart, liver and kidneys were subjected to careful microscopic examination, a few hours after death. The blood from the heart presented the characteristic golden colored areola, when dropped upon bibulous paper, and under the microscope the colored blood corpuscles presented a crenated and stellate appearance. No animalculæ or sporules, or vegetable organisms were observed, even under the highest powers of the microscope. Numerous vibrios were observed in the black vomit taken from the stomach after death. The muscular structures of the heart presented a pale, yellowish-brown, flabby appearance and contained much free oil in the form of globules. The liver presented the characteristic yellow color, and contained much oil, deposited in the form of oil globules in and around the secretory cells of this organ. The kidneys presented a yellowish-brown color, and the excretory tubes were filled with yellow granular matter. I injected, by means of a syringe, (the nozzle of which was plunged through the skin) into the cellular tissue of an active dog both black vomit from the stomach and fluid blood from the cavities of the heart, and also applied the black vomit to the mucous membrane of the eyes. The animal also swallowed a portion of black vomit and blood, after fasting. No ill effects were observed and the animal remained apparently well and active.

In the case of Newton Simpson, who was attacked on Oct. 11, 1871, and died with urinary suppression and profuse black vomit, on the ninth day of the disease, the textures of the body, divided by the knife, presented the bloody appearance usual in such cases of yellow fever; urea was detected in the blood, brain and liver. The heart contained in all its cavities dark fluid blood, which gave an acid reaction at the time of the post-mortem examination, ten hours after death. Under the microscope, many of the blood corpuscles presented a crenated, wrinkled appearance, others were swollen, while others again presented the usual appearance.

I could after careful examination, with high powers, detach no animalculæ, or simple vegetable or animal forms, although the search was conducted with great care, with powers of various degrees, from one-fourth to one-eighteenth of an inch objectives, and the highest oculars. The blood was fluid, with little or no fibrin, and no fibrinous clots; the fibrin was in so small an amount, and in such a soft and unstable condition that it was impossible to determine the quantity. Specific gravity of blood 1046.

1,000 parts of blood from the cavities of the heart contained:

Water	852.70
Solid residue	147.30
Fibrin	Traces.
Saline constituents	9.80

From experiments which I have conducted, it appears that the specific gravity of the blood in the cavities of the heart after death from yellow fever is less than that contained in the large blood vessels. This appears to be due to the fact that the heart continues to circulate the blood, or to force it through its cavities during the last moments of life, the red blood corpuscles are arrested in the capillaries, and only the more tenacious constituents of the blood are returned to its cavities during the last moments of life. In this case, the spleen was somewhat enlarged and softened, and its "pulp" consisted of numerous colored corpuscles, oil globules and granular masses; but no living animalculæ or vegetable forms were observed. Blood corpuscles of spleen not specially altered in appearance. Oil globules abundant in spleen.

The reaction of the blood from the liver was acid, and at the end of twenty-four hours numerous prismatic crystals, of the triple phosphate of lime, magnesia and ammonia formed in the fluid oozing from the cut surface of the liver. The blood of the liver contained numerous oil globules, but no specific animalculæ or vegetable organisms.

The results of the microscopic examinations of the structures of the heart, liver and kidneys were similar to those detailed in the report of the case of John Allen.

In the case of Jacob Siegarist, age 26, native of Germany, who was attacked Oct. 12, 1871, and who suffered with well marked yellow fever, characterized by suppression of urine and alkalin black vomit, and who died on the seventeenth day of the disease, urea and bile were found in the blood and in all the organs, brain, liver, spleen and heart. At the necropsy, performed three hours after death, both cavities of the heart contained warm fluid blood, which gave a slight acid reaction to litmus blue paper and upon standing coagulated, forming a soft gelatinous clot which possessed no contractile power. The aorta, vena cava and pulmonary veins in like manner contained warm fluid blood, which coagulated imperfectly upon standing.

EXAMINATION OF FLUID BLOOD FROM CAVITIES OF HEART THREE HOURS AFTER DEATH.

The blood after its abstraction coagulated, forming a loose clot; the fibrin, however, dissolved and no serum was separated. Under the microscope the blood corpuscles presented no special alterations; when spread upon the glass slide, and also during the coagulation of the blood, they rapidly agglomerated together, forming rouleaux, as in inflammation in the blood of the horse. The running together of the colored corpuscles was as rapid and as complete as in cases of well marked inflammation.

It was impossible to determine the weight of the fibrin, or to collect pure serum, free from colored blood corpuscles and the coloring matter of the red corpuscles, as the fibrin rapidly dissolved after its partial separation. The fibrin was in very small amount, and apparently not one-thirtieth of the usual proportion. Specific gravity of blood from cavities of heart, 1047.

1,000 parts of blood from cavities of heart contained:

Water		821.57
Solid residue 178.43	Organic matters, colored corpuscles, albumin, urea, extractive and fatty matters, etc.	170.59
	Fixed saline constituents	7.84
	Fibrin	Traces

Three hours after death, specific gravity of blood from vena cava 1062. Blood fluid and warm when drawn; coagulated, forming a very loose coagulum, which did not inclose the whole amount of colored corpuscles. This coagulum gradually dissolved, and it was impossible to determine the amount of fibrin or to obtain clear serum free from colored corpuscles. Reaction of blood slightly acid.

1,000 parts of blood contained:

Water		775.00
Solid residue 225.00	Organic matters, colored blood corpuscles, albumin, urea and ammonia.	217.31
	Fibrin	Traces
	Saline constituents.	7.69

It will be observed that the blood in the vena cava contained more solid matter than that in the cavities of the heart; it would appear that the returning current of blood from the capillaries and venous system, owing to the tendency to coagulation and congestion, contained more solid matters than the blood of the heart.

No vegetable or animal forms were discovered in the blood.

Yellow fever blood exposed upon my table, in capsules and bottles for days and weeks, and examined microscopically from time to time, developed no special forms which could be referred exclusively to yellow fever. Only the simpler forms of animal and vegetable life made their appearance, which might be developed in any other similarly constituted albuminous fluid.

In a fatal case of yellow fever, occurring in one of my wards in the Charity Hospital during the month of October, 1871, in a stout young man, aged 25, who from the results of the post-mortem examination, had evidently suffered with malarial fever, incipient cirrhosis of the liver and granular degeneration of the kidney, before the supervention of yellow fever; death occurred after an illness of ten days; the disease was complicated during its progress by the supervention of pericarditis, pleuritis and pneumonia. The urinary secretion was abundant until near the close of life, and the urine contained blood corpuscles and the coloring matters of the blood. The cavities of the heart were filled with loosely coagulated blood. Portions of the lungs were infiltrated with blood, as if hemorrhage had taken place in and around the textures of the lung. Louis, in his "Anatomical, Pathological and Therapeutic Researches on the Yellow Fever of Gibraltar" of 1828, has shown that the exudation of blood into the pulmonary tissue was frequent, while the inflammation of that tissue was rare in subjects who had died of yellow fever. He found the lungs entirely natural in three subjects only.

Black spots, and masses of the same color, more or less impermeable to the air, were found in these subjects usually of a brownish black, rarely of a crimson hue; they were more or less concentrated, and occupied a variable space at the exterior or in the interior of the lung, and in some cases they were found only in the lower lobe. The black or blackish masses which existed in the lungs of six individuals contained no air, had no granular aspects as in the hepatized lung of pneumonia and presented no traces of organization; usually they could be easily broken down, in some cases yielding by pressure; the blood of which they were almost entirely composed, and the pulmonary parenchyma remained apparently of its natural consistency. In this case complicated by the suppuration of inflammatory disease, the fibrin of the blood was not diminished to the extent usual in yellow fever. The pre-existing effects of malarial fever were evident in the masses of black pigmentary matter, derived from the hematin of the blood, scattered through the lobuli of the liver, but chiefly accumulated in the peripheral portions, in and around the portal veins and portal capillaries.

In uncomplicated yellow fever these altered masses of hematin are uniformly absent from the structures of the liver, their place is supplied by oil globules; and it would appear that in this disease the albuminous constituent of the blood and tissues is chiefly altered, the altered albumin appearing in abundance in the urine, while in malarial fever the colored blood corpuscles are chiefly attacked, the coloring matter being deposited in various organs, but in the acute stages of uncomplicated malarial fever, albumin rarely, if ever, appears in the urine.

The results of my investigations upon the occurrence of pigment in the blood and in certain organs, as the liver, spleen and kidneys, in the capillaries of the brain, in persistent intermittent and remittent malarial fevers, in chronic malarial poisoning, correspond with those of Meckel, Virchow, Heschl and Platner, and more especially with the researches of Frerichs. Not only is the spleen enlarged and the color of the liver altered to a steel gray, blackish slate and chocolate or bronze color, in individuals who die from the effects of marsh poison, but this change of color may extend to other organs as the kidneys and brain. Careful microscopic examinations of sections of the various organs have shown that these changes of color are clearly referable to the formation and accumulation of pigment matter resulting from destructive and abnormal changes of the colored blood corpuscles. While from the anatomic structure of the spleen, as well as from the great congestion which it undergoes during the cold stage of malarial fever, it is without doubt true that a large portion of this pigment is formed in the sinuses of the spleen and passes from this organ into the portal veins, and that part of it remains impacted in the capillaries of the liver, while the rest passes through these capillaries, and is carried into the general circulation; at the same time it must be admitted that under the action of the malarial poison, the transformation of the red matter of the blood into black pigment may take place everywhere throughout the vascular system, and also external to it, but more especially in the liver and kidneys. If fine sections of the malarial liver be made with Valentine's knife, and examined under comparatively low magnifying powers (one and a half to one inch), as I have done in a large number of subjects, accumulations of pigment will be observed in the capillary network of the portal and hepatic veins, either uniformly distributed or limited to certain regions, sometimes deposited chiefly in the interlobular veins, forming black margins to the lobules of the liver; or more uniformly, extending from the circumference of the lobules halfway to their center, or penetrating as far as the beginning of the hepatic veins. The hepatic cells, as in the case under consideration, remain exempt from the deposits of black coloring matter.

That portion of the pigment originating in the spleen and passing through the capillaries of the liver, enters the general circulation and may be deposited in various organs as the brain, kidneys and lungs, inducing morbid symptoms varying in character and degree with the organ whose capillary circulation may be especially retarded by the arrest of the pigment particles. The larger particles remain impacted in the capillaries of the portal vein and obstruct the circulation of the blood through these vessels, causing various derangements in the secretion of the liver, and in some cases extensive capillary stagnation of the blood in the roots of the portal vein, attended with exhausting hemorrhages from the gastro-intestinal mucous membrane, of an intermittent character, profuse diarrhea, vomiting, serous effusions into the peritoneal sac and, finally, from the obstruction of many of the smaller branches of the portal vein, chronic atrophy of the liver, with its distressing and fatal consequences.

Even by simple inspection of the brain we may form some idea, not only as to the cause of the alteration from the color of health, but also of the amount of pigment which has passed unarrested through the vessels of the liver and lungs. The mechanical interruption to the circulation of the brain thus induced, not only gives rise to a peculiar train of symptoms, but may also cause the rupture of the small vessels and the formation of numerous capillary apoplexies, as has been shown so clearly by Meckel, Platner and Frerichs.

Frerichs has shown, and I have upon many occasions confirmed the accuracy of his investigations, that the large pigment granules and cells which enter the kidneys along with the arterial blood, not unfrequently become impacted in the capillary coils of the Malpighian bodies, and by altering the pressure of the blood give rise to derangements in the secretion of urine, which exercise a powerful influence over the further progress of the disease. Albuminuria, fibrinous casts, pigment masses and granules, blood corpuscles characteristic of the urine in malarial hematuria should to a large extent be referred to the effects of the pigment particles arrested in the capillaries of the kidneys. Hemorrhage, congestion, exfoliation of the excretory cells of the tubuli uriniferi, fibrinous exudation into the excretory tubes, and even complete suppression of the urinary secretion may occur in such cases. Destruction of the colored blood corpuscles may in like manner occur directly in the capillaries of the kidneys in malarial fever, and the pigment thus formed may give rise to a similar train of symptoms.

As far as my observations have extended, no such changes take place in the colored blood corpuscles and various organs in yellow fever—they are not destroyed in the various organs, neither is there a special diminution of these important elements, and all the organs and tissues are free from the presence of pigment granules in uncomplicated yellow fever.

If, however, as in the preceding case, the yellow fever occurs in one who has previously suffered with malarial fever, or if the disease supervenes upon yellow fever, then the pigment matter may be present to a greater or less extent, especially in the liver and spleen.

The result obtained by actual chemic analysis and careful microscopic research, viz.: that the colored blood corpuscles are not specially destroyed in yellow fever, as is the case in malarial fever, is still further sustained, by the well-established fact that, in general, convalescence is rapid and complete, and attended with a speedy restoration of all the healthy functions. Such a rapid and complete restoration would be impossible if the colored blood corpuscles were destroyed to any great extent in yellow fever.

During the latter months of 1870, when 587 deaths from yellow fever, were officially reported by the authorities of New Orleans, although the number was probably greater, as 445 deaths were during the same period (August, September, October, November and December), referred to the various forms of malarial fever, I instituted in like manner careful chemic and microscopic examinations of the urine, blood and black vomit of the various organs in yellow fever, and the results were similar in all respects to those recorded at length in the preceding pages; we shall for the sake of brevity simply allude to some of the more general results.

The type of the disease was severe and I saw cases in which the defibrinated blood oozed from leech bites, from the ears and from the gums and mouth; some cases suffered with profuse hemorrhage from the bowels before death. The bodies underwent putrefaction a very short time after death, having emitted during life a most disagreeable, foul yellow fever odor. The black vomit contained uniformly colored blood corpuscles variously altered; also mucous epithelium from the gastric mucous membrane; sometimes various matters, as medicine, food and drink taken by the patient; oftener numerous vibrios and plants resembling the torula and sarcinæ were present, but I was unable to detect any specific vegetable forms characteristic of yellow fever. The urine contained albumin and in the fatal cases was almost uniformly suppressed. In the worst cases the urine consisted only of a yellowish albuminoid fluid discolored by bile, containing little or no urea, but loaded with excretory cells and granular fibroid casts of the tubuli uriniferi. The lightest colored urine was frequently characteristic of the severest cases.

As a general rule, the blood changed to the arterial hue upon exposure to the atmosphere, and the cut and exposed muscles presented a beautiful scarlet color.

In some cases golden-colored heart clots of small size and slight consistency, composed of yellow laminated fibrin were observed in the cavities of the heart, and in those cases which had suffered with black vomit. Blood drawn from the cavities of the heart, after death from yellow fever, and carefully examined under the microscope presented swollen and stellate blood corpuscles. This stellate condition of the blood corpuscles appears to be very common in the blood of yellow fever, and even in that drawn from the veins during life. When a drop of yellow fever blood is allowed to fall upon white bibulous paper, the red corpuscles occupy the original area of the drop, and are surrounded by a golden circle of the serum colored by bile. In any fatal case occurring during the fall of 1870, which fell under my observation, the serum was of a bright golden color from the presence of bile.

The nervous symptoms characteristic of this disease were referred, in great measure at least to the retention of the biliary matters in the blood, to the suppression of the function of the kidneys and the retention in the blood of the urinary constituents, especially of the urea in the blood.

The chief causes of death in these cases of yellow fever appeared to be the direct action of the febrile poison upon the nervous system and blood, depressing and deranging the one and rendering the other unfit for the proper nutrition of the tissues; the suppression of the functions of certain organs, as the kidneys and liver and the retention in the blood of the excretions normally eliminated by these organs. Blood loaded with bile and urea is evidently unfit for the maintenance of healthy nutrition and of the vital acts.

The hemorrhage from the gastric mucous membrane appeared to have been due to several causes, as the direct irritant effect of the poison upon the gastric mucous membrane, the destruction of the fibrin of the blood, the physical and chemical alterations of the albumin, and the irritant and dissolvent and *excoriating* effect of the carbonate of ammonia, resulting from the decomposition of the urea.

The black vomit in yellow fever, as shown by the observations upon numerous specimens obtained during life, and taken from the stomach after death, consists of altered blood corpuscles, the epithelial cells of the stomach, mucus corpuscles, various matters received into the stomach, as food, medicine, water, etc., serous exudations, acids of the gastric juice (acetic, hydrochloric and phosphoric), urea, carbonate of ammonia, and various forms of animal and vegetable life of simple organization.

After careful examinations of numerous specimens of black vomit, we failed to detect any forms which could be considered as characteristic of this fluid; which may not be developed in albuminous fluids exposed to the action of the atmosphere, at all times, whether yellow fever be present or absent.

The careful microscopic examination of the blood with the highest powers, was equally fruitless in disclosing any forms of animal or vegetable life which could be considered as active in the production of yellow fever, or as invariably accompanying its manifestations.

We have established by the preceding observations:

1. The blood in yellow fever often if not always contains abnormal amounts of *urea* and *bile*.

2. The presence of the urea and bile in the blood is attended with certain nervous disturbances.

3. It is probable that the extreme slowness of the pulse which characterizes many cases of yellow fever, after the subsidence of the first stage of febrile excitement, is due to the presence of the bile and urinary constituents in the blood, as well as to the anatomic lesions of the heart.

4. The fibrin is greatly diminished in the blood of yellow fever.

5. The passive hemorrhages and capillary congestion in yellow fever are without doubt largely dependent upon the diminution of the fibrin.

6. The colored blood corpuscles are not specially diminished in yellow fever; and in this respect the disease differs widely from malarial fever.

Case of yellow fever engrafted on malarial fever; suppression of urine; black vomit; hemorrhage into gall bladder. Francis Dubusé; harness maker, age 32, native of France. Admitted Oct. 4, 1874, to Charity Hospital with intermittent fever; discharged October 9. After his discharge moved across the river to Gretna and followed the occupation of milkman. Some time, however, in the month of November, returned to the city and resided in Chartres Street in a neighborhood in which cases of yellow fever had occurred.

Entered Charity Hospital December 4. Had been sick with pain in the head, back and extremities for six days and had thrown up black vomit before entering Charity Hospital. Condition on entrance: deep yellow (jaundiced) color of skin and conjunctiva of eyes; sluggish capillary circulation; slow pulse; nausea; vomiting black vomit; temperature but slightly elevated above the normal standard. Urine of yellow color, turbid from the presence of urinary casts and cells from the urinary tubes and granular matter. Urinary casts as soon as the albumin coagulated by heat, presented a golden color. Diagnosis: yellow fever. The urinary excretion progressively diminished in amount, and toward the close of life, during the last thirty hours, was entirely suppressed. Died December 9, on the ninth day of the disease.

Post-mortem six hours after death. Exterior, head, trunk and extremities pale; surface of entire body of a golden color; the dependent portions being mottled of a purplish color. Black vomit issuing from corners of mouth. Head: blood vessels of brain congested with dark blood. Thorax: lungs normal; lower portions of darker color than upper portions, from settling of blood. Heart: yellow, flabby, softened muscular textures readily crushed between the fingers, and under the microscope the fibers of the heart presented the changes characteristic of yellow fever, the transverse striæ being indistinct, and numerous oil globules and yellow granular matter being deposited within and around the muscular fibers.

Abdominal cavity: mucous membrane of the stomach, of a deep purple color; with deep ecchymoses. Under a low magnifying power the ruptured mouths of the small blood vessels were clearly seen. The stomach contained several ounces of thick black vomit resembling putrid blood. Reaction of black vomit neutral. Specific gravity of black vomit 1032. Upon analysis, 1,000 grains of black vomit yielded 70 grains of coagulable albuminoid matters. After the removal of the albuminoid and coloring matters of the blood from the black vomit, the remaining liquid contained in addition to phosphates, sulphates and chlorids of the alkalies and alkalin earths, urea and salts of ammonia. Under the microscope, the morphologic elements of the black vomit were found to be colored blood corpuscles, mucus, corpuscles and cells from the mucous membrane of the stomach. No spores or vegetable organisms were observed. Upon standing, vibrios made their appearance in considerable numbers; but no fungoid growths were developed even at the end of forty-eight hours. The temperature appeared to be too low for the rapid development of the fungi. It is, however, worthy of note that the yellow fever had attacked this patient in the winter, December 1.

Liver: This organ presented on the surface a dark purplish mottled color; and upon a superficial examination presented nothing abnormal in color, except an increase in the depth of the normal shade. When sections of the liver were made, carefully washed and examined they presented a distinct and characteristic mottled color, many of the lobuli being of the peculiar yellow color of yellow fever, and others presenting a variegated yellow and deep bronze color. Careful microscopic examination revealed the fact that the yellow portions were loaded with oil globules and yellow granular matter as in yellow fever, and that the more deeply colored portions contained numerous pigment particles as in malarial fever. The mottling of the usual yellow fever deposits, was due to the deposit of pigment particles in and around the portal capillaries during the preceding attack of malarial fever. Under the microscope the liver cells were found to be loaded with oil globules as in yellow fever; dark pigment particles were also scattered through the structures of the liver, but existed in greatest numbers within and around the peripheral or portal capillaries of the lobuli. The peculiar appearance of the liver in this case, was clearly the results of the combined effects of yellow fever and malarial fever; the latter having preceded the former.

Gall bladder: the gall bladder was distended with a dark fluid of a brownish and reddish black color, and upon a superficial examination resembled this viscus in malarial fever. When the bile was poured out it resembled in appearance dark liquid blood. Specific gravity of the fluid from the gall bladder 1021.5; reaction neutral. Amount of fluid in the gall bladder 1,600 grains. Heat and nitric acid caused coagulation; coagulum of a dark mahogany color like that always formed in liquid blood under the action of heat and nitric acid.

Under the microscope, the fluid from the gall bladder was found to contain numerous altered colored blood corpuscles and epithelial cells of the mucous membrane of the gall bladder. The mucous membrane of the gall bladder was of a deep purple congested color, resembling in all respects the deeply congested and purple mucous membrane of the stomach. Amount of coagulable albuminoid matter in 1,600 grains of bloody liquid from the gall bladder, grains 44.70. Nitric acid produced no change of color, similar to that caused by the coloring matter of the bile; numerous tests also repeatedly and carefully applied failed to show the presence of the biliary acids. The reagents simply caused coagulation of the albumin, as in ordinary liquid and diluted blood. The filtrate obtained after the coagulation and removal of the albuminoid elements of the blood was found to be loaded with urea. The liquid therefore found in the gall bladder was not *bile* but *altered liquid blood*, similar to that poured into the stomach and called black vomit.

We have in this case and in the preceding facts, a striking and important illustration of the sources of error in the investigation of the pathologic anatomy of yellow fever. Superficial observers here and elsewhere have proclaimed that yellow fever had no distinctive pathology. Such assertions possess no value unless based upon the most careful and thorough microscopic and chemic investigations.

Kidneys: these organs presented a yellow color resembling that of the heart. When sections of these organs were made with Valentin's knife and carefully examined under the microscope, the Malpighian corpuscles and tubuli uriniferi were found to be loaded and impacted with desquamated cells, oil globules and yellow granular matter.

Yellow fever engrafted on intermittent fever, suppression of urine; black vomit; death; hemorrhage into gall bladder. E. Johnson, age 27, native of New York, laborer. Came to New Orleans in January, 1874. Went from this city to work on jetties at the mouth of Mississippi River. Entered Charity Hospital Nov. 20, 1875. Patient stated that he had been suffering with intermittent fever for one month. Walked about the ward until the evening. I saw him the next morning, November 21. Has rapid pulse and high fever. Quinin was administered freely but without any effect upon the fever. November 22, fever continues; patient very restless; eyes injected; tenderness over region of liver, epigastrium and small of back over the kidneys. Has been vomiting. Quinin repeated, but without any beneficial effects. November 23, fever declining; great capillary congestion of face, neck and extremities; yellow tinge of surface; very restless and delirious; injected eyes; urinary excretion greatly diminished. November 24, heat of body almost normal, but capillary congestion intense and jaundice increasing. Urinary excretion greatly diminished and in fact almost wholly suppressed; delirium; has vomited dark "coffee ground" matter. November 25, restless and delirious; pulse 68; respiration 25; temperature of axilla 95 degrees. Head, trunk and extremities feel cool. Great capillary congestion of surface which presents a purplish and mottled appearance, with a distinct golden color; complete suppression of urine. Upon careful examination the bladder is found to be entirely empty; great prostration of muscular and nervous systems; patient delirious and restless. During the past two days it has been impossible to arouse the patient or to obtain from him any coherent answer; 6 P.M., pulse 68; respiration 28; temperature of axilla 95.5 degrees. The patient died one hour after this observation.

Necropsy thirteen hours after death: Exterior, trunk and limbs full, round and plump; skin yellow; dependent portions of head, neck, trunk and extremities present a purplish mottled appearance. Thorax: heart, yellow and softened; weight nine and five-eighths ounces. Transverse muscular striæ indistinct; oil globules and yellow granular matter deposited in large amount, within and around the muscular fibrillæ of the heart. Lungs normal; dependent portions congested. Abdominal cavity: liver yellow and enlarged. Under the microscope, sections of the liver, with Valentin's knife, presented the characteristics of the yellow fever liver; immense numbers of oil globules of various sizes, and yellow granular matter deposited within and around the cells and capillary networks of the liver. The liver also contained pigment particles, resulting from the preceding attack of malarial fever.

Gall bladder: The gall bladder contained 421.4 grains of thick, deep green, almost black, tenacious bile, which poured with difficulty and could be drawn out into long strings. When heated the whole mass coagulated firmly, resembling in this respect albumin or liquid blood, colored by biliary matter. Under the microscope the bile was found to contain an immense number of desquamated cells from the mucous membrane of the gall bladder, also colored blood corpuscles and masses of hematin. Mucous membrane of gall bladder greatly injected and discolored, of a deep purplish and greenish color. When held up to the light a dense network of deep purple and black congested capillaries and blood vessels were everywhere discoverable. Solid coagulable matters in 421.4 grains of bile, grains 77.4; solid matters in 1,000 parts of bile, 183.67. The contents of the gall bladder were so thick and tenacious that it was impossible to introduce them into the ordinary specific gravity bottle.

It is evident that hemorrhage had taken place into the gall bladder. Stomach distended with black vomit, resembling dark fluid blood; the intestines also contained much black vomit, of a character similar to that found in the stomach. The great depression of temperature, about 4 degrees F. below the normal standard, which characterized the last twenty-four hours of life may have been due to the profuse hemorrhage into the stomach and bowels. Under the microscope the black vomit from the stomach presented nothing more than mucus corpuscles, desquamated cells from the gastric mucous membrane, broken capillaries, and immense numbers of colored blood corpuscles. I observed no vegetable nor animal organisms. Their absence appeared to be due chiefly to the depressed temperature of the surrounding atmosphere. Kidneys engorged with blood; when divided and washed, they presented a yellow color like that of the liver. Weight, seven and one-eighth ounces. Chemic analysis showed a great increase of oil in the structures of the kidneys, as well as of the heart and liver. When sections of the kidneys were made with Valentin's knife and carefully examined under the microscope, the Malpighian corpuscles and tubuli uriniferi were found to be impacted (literally stuffed) with detached cells, oil globules and yellow granular matter. The urinary bladder contained only two fluid drachms of urine, which represented the whole amount of urine excreted by the patient, during the last twenty-four hours of life. The urine contained albumin and numerous casts of the tubuli uriniferi, filled with yellow granular matter and oil globules, also cells from the pelvis and ureter, and from the mucous membrane of the bladder. The urine contained, also, little or no bile and presented a yellowish red color.

In this case the disease was contracted at the jetties near the mouth of the Mississippi River, where fever prevailed to a limited extent among the laborers, late in the season. One or more barges upon which the workmen slept at night became infected, and it was found necessary to destroy them by fire.

CHAPTER VII.

EFFECTS OF JAUNDICE UPON THE FREQUENCY OF THE PULSE.

Case 1.—Malarial fever, dysentery and jaundice. A. F., age 32; native of Germany. Entered Charity Hospital Dec. 29, 1875. Had had intermittent fever. Says that he has been sick with intermittent fever and dysentery for fourteen days. Upon entrance presented a pale sallow, bloodless hue, and suffered from painful discharges from the bowels, of mucus and blood at regular intervals. On January 1, the patient showed a distinct yellow tinge of the skin and conjunctiva of the eyes, which gradually increased to the most intense jaundice in the course of ten days. The urine was heavily loaded with bile, but free from albumin. The pulse which had been rapid and feeble, ranging from 100 to 120, became much slower after the supervention of the jaundice. The effect of the bile in the blood upon the frequency of the pulse is shown in the following table:

Date.	Pulse.		Resp.		Temp.	
1875	M.	E.	M.	E.	M.	E.
Jan. 13	....	70	....	....	....	99 °
14	....	70	....	32	....	101
15	....	80	....	36	....	100
16	70	80	28	32	99 °	101
17	100	100	24	32	101	104
18	80	92	32	32	98	102
19	70	92	24	32	99.5	103.5
20	72	80	28	36	99.5	103.5
21	80	88	20	20	98.5	103.5
22	76	84	24	24	99.5	101
23	76	72	32	24	99.5	100.5
24	72	68	28	28	99.5	100
25	....	80	....	20	....	99.5
26	72	72	36	40	97.5	98.5
27	72	76	40	36	99.5	99
28	60	72	36	32	98.5	100
29	72	64	32	32	99	99.5
30	60	64	28	36	99.5	100
31	....	68	....	32	....	99
Feb. 1	68	68	40	32	99.5	99.5
2	68	....	32	....	98.5	....

The periodic variations of the temperature in this case were regarded as the result of the action of the malarial poison. It will be observed that in this case after the supervention of jaundice, the pulse was only 100, with a temperature of 104 degrees F., and on January 20, the pulse was only 80, with a temperature of 103.5 degrees F. On the 30th the pulse was only 60, notwithstanding the continuance of the dysentery, with frequent discharges from the bowels.

The treatment of this case consisted mainly in the rigid adherence to milk and rice diet and beef tea, and the employment at regular intervals of a mixture of quinin, subnitrate of Bismuth and Dover's powder; in the proportion of 3 grains each of quinin and Dover's powder, and 10 grains subnitrate of bismuth, every three, four or six hours. These powders were also alternated with a simple mixture of syrup of morphia and subnitrate of bismuth, or with a mixture composed of equal parts of the tinctures of kino catechu, opium, camphor, capsicum and Peruvian bark, a teaspoonful in a wineglass of water every three or six hours. Port wine and alcoholic stimulants in small quantities were also found to be beneficial. This was a protracted and tedious case, but the patient was finally restored to health and was discharged in the month of March.

Case 2.—Malignant scarlet fever attended with jaundice; slow pulse, recovery. Adam Grethiel, age 19; native of New Orleans. Entered Charity Hospital Feb. 11, 1876. February 11, deep scarlet eruption; where the eruption is not very great the skin presents a yellowish tinge. Conjunctiva of eyes yellow. Mucous membrane of throat and tonsils swollen and ulcerated. Tongue dry and red; patient swallows with difficulty; hemorrhage from the mouth and gums; wild delirium; necessary to confine the patient to bed. Has been sick about one week. Abscesses are beginning to form near the elbows and at the extremities of the fingers; P.M., pulse 100, respiration 48, temperature 103.5. February 12, A.M., pulse 100, respiration 36, temperature 102; P.M., pulse 108, respiration 28, temperature 103. Desquamation is beginning, and as the scarlet color diminishes, the deep yellow color becomes more manifest. February 13, A.M., pulse 100, respiration 36, temperature 100; P.M., pulse 124, respiration 36, temperature 103.5. February 14, A.M., pulse 88, respiration 28, temperature 100; P.M., pulse 100, respiration 32, temperature 99. The pulse continued to decline in frequency and on the 16th was 72, with a respiration of 24 and temperature 101. It would consume too much space to present the numerous details relating to the pulse, respiration and temperature in this case; the pulse, however, continued to grow slower notwithstanding the condition of the throat and the formation of abscesses and on the 19th the pulse was 68, respiration 24, temperature 101; on the 22d, pulse 64, respiration 20, temperature 100.5. The patient finally recovered entirely.

Case 3.—Pneumonia, with intense jaundice; albumin and bile present in the urine, chlorids absent; frequency of the pulse diminished by the bile in the blood. John T. Webb, age 37, native of Maryland. Has resided in New Orleans for eighteen months. Entered Charity Hospital January 8 in a restless sleepless state and apparently suffering from alcoholism. Patient states that he "had been drinking very hard before entering the hospital." The bowels were opened with the compound cathartic pill (U. S. P.), and the nervous agitation was measurably relieved by bromid of potassium in combination with liquor ammoniæ acetates. On January 10, the patient complained of pain in the right side, with cough and oppression of breathing. Dullness upon percussion, with crepitation over lower and middle lobe of right lung. Pulse and respiration greatly accelerated, temperature elevated. January 11, evident marks of pneumonia of right lung; the conjunctiva of eyes and skin present a distinct yellow tinge. January 12, jaundice well marked, high fever, pulse decreasing in frequency, falling from 130 to 112. Bile and albumin present in urine, chlorids entirely absent. There is no hepatic tenderness.

January 13, cough, pneumonic sputa, headache, pain in back and limbs; no hepatic tenderness, albumin and bile in urine, chlorids absent. Skin of a golden yellow color, conjunctiva of eyes yellow. Patient complains of great depression of spirits and restlessness. At 8 A.M., pulse 100, temperature of axilla 101. 14th, A.M., pulse 128, respiration 32, temperature 99; P.M., temperature 102. 15th, P.M., pulse 100, respiration 32, temperature 99.5; P.M., temperature 100.5. 16th, A.M., pulse 100, respiration 20, temperature 99.5; P.M., pulse 109, respiration 32, temperature 99. 17th, A.M., pulse 108, respiration 28, temperature 100; P.M., pulse 112, respiration 24, temperature 101. 18th, A.M., pulse 88, respiration 24, temperature 98.5; P.M., pulse 100, respiration 32, temperature 101. 19th, A.M., pulse 100, respiration 28, temperature 100; P.M., pulse 100; respiration 24, temperature 102. 20th, A.M., pulse 100, respiration 32, temperature 99.5; P.M., pulse 100, respiration 28, temperature 101. 21st, A.M., pulse 84, respiration 32, temperature 98.5; P.M., pulse 88, respiration 32, temperature 101. 22d, A.M., pulse 80; respiration 32, temperature 98.5; P.M., pulse 100, respiration 40, temperature 101.5. 23d, A.M., pulse 72, respiration 32, temperature 98.5; P.M., pulse 92, respiration 32, temperature 101.

Convalescence was tedious in this case, but the patient was discharged on the 23d. In the intensity of the jaundice and in the presence of albumin in the urine this case upon a superficial examination resembled yellow fever. The effect of the bile in the blood on the action of the heart will be still further evident by comparing the variations of the pulse and temperature in the three preceding cases, with the three following:

Case 4.—Pneumonia of right lung, rapid pulse, rapid respiration and elevated temperature—fatal issue. Thomas Gegan, age 20, native of Ireland, baker. On Feb. 10, 1876, went to sleep on cold ground, and on awakening suffered with severe pain in the right side. Entered Charity Hospital Feb. 17, 1876. He says that he has been sick with fever and severe pain in the right side for five days. February 17, evening, pulse 140, respiration 48, temperature 105.5 F. Some pain in right side, with cough but little expectoration; sputa contains much pus mixed with the rusty colored fibroid matter. Crepitation and dullness over right lung and over lower lobe of left lung. A cavity is evidently forming in the infraclavicular region of the right lung. Percussion gives the cracked-pot sound, and auscultation reveals amphoric respiration. The purulent matter comes from this cavity in great measure. February 18, A.M., pulse 140, respiration 44, temperature 105.5; P.M., pulse 128, respiration 40, temperature 106. No albumin in urine, bile present, chlorids wholly absent; specific gravity 1010. 19th, A.M., pulse 140, respiration 36, temperature 104.5; P.M.. pulse 140, respiration 36, temperature 104.5. 20th, A.M., pulse 146, respiration 48, temperature 106; P.M., temperature 103.5. 21st, A.M., pulse 140, respiration 36, temperature 105; P.M., pulse 140, respiration 24, temperature 103.5. 22d, A.M., pulse 148, respiration 28, temperature 105; P.M., pulse 144, respiration 24, temperature 104.5. 23d, A.M., pulse 120, respiration 28, temperature 103.5. On the 20th the patient became delirious, with occasional lucid intervals and died on the 23d.

We might greatly multiply such cases, but they have been illustrated in the first volume of my "Medical and Surgical Memoirs."

Case 5.—Chronic malarial poisoning and intermittent fever, illustrating the rapid circulation in this disease. J. Hogan, age 29, native of New Orleans, carpenter, admitted to Charity Hospital Oct. 21, 1875; has been working on Baker

River, Louisiana, and has suffered with intermittent fever for two months. October 23, sallow anemic complexion, presenting a dusky greenish-yellow color; has fever as in chronic malarial poisoning. A.M., pulse 122, respiration 32, temperature 103.25; P.M., temperature 102. 24th, A.M., pulse 128, respiration 36, temperature 103; P.M., temperature 101.5. 25th, A.M., pulse 130, respiration 24, temperature 101.5; P.M., temperature 101. 26th, A.M., temperature 100; P.M., temperature, 102. 27th, A.M., pulse 100, respiration 24, temperature 101.5; P.M., temperature 102. On October 25 I administered to this patient, in addition to the full doses of quinin, tincture of digitalis. The reduction of the frequency of the pulse was marked and rapid. In this case with a temperature of 101.5, the pulse reached 130 beats per minute. The urine was high colored, but contained no albumin.

Case of circumscribed pneumonitic pleuritis; rapid pulse; fall of pulse after effusion into pleura and lungs. George Allen, sailor, entered Charity Hospital Jan. 18, 1875. January 19, pain in left side; hurried respiration; pleuritic friction sound and crepitant râles in lower and middle lobes of left lung; delirium. A.M., pulse 150, temperature 103.6. 20th, A.M., pulse 120, respiration 45, temperature 101.5. 21st, A.M., pulse 112, respiration 36, temperature 99. The urine was loaded with urates but contained no chlorids. Albumin absent. The dullness upon percussion increased, effusion has taken place and with it the temperature has fallen and the pulse has become slower.

January 22, A.M., pulse 72, respiration 20, temperature 97.7. 23d A.M., pulse 72, respiration 25, temperature 97.8. The delirium and mental aberration continued for four days and was followed by two days of sleep and drowsiness. Patient recovered.

It will be observed that in this case of circumscribed pleuro-pneumonia the pulse was 150, with a temperature of 103.6 degrees.

Case 6.—Typhoid fever and double pneumonia, rapid pulse, high temperature; death on thirteenth day of disease. Thomas Feverson, age 22, native of Norway, sailor. Has been in New Orleans during the past five months; came direct from Europe. Entered Charity Hospital Feb. 1, 1876. At time of entrance had fever, which was wholly uninfluenced by quinin which was daily given for four or five days without any manifest effect. February 4, P.M., skin hot and dry; dichrotic pulse, 160 per minute, respiration 28, temperature of axilla 103.5. Dullness upon percussion over lower lobes of both lungs, with crepitant râles and rusty colored sputa. Epistaxis, delirium, subsultus tendinum. Tenderness in right iliac region, with some gurgling; bowels slightly tympanitic; stools not frequent but loose. Passed excrement and urine in bed; sordes on teeth; tongue red and dry, not furred. The tympanitis has been greatly relieved by turpentine stupes over chest and abdomen. Chlorids absent from urine; high color. Albumin present in small quantities in urine. February 5, ninth day of disease, morning, pulse 100, respiration 24, temperature of axilla 100. February 6, tenth day of disease, morning, pulse 100, respiration 20, temperature 100; P.M., pulse 112, respiration 32, temperature 104. 7th, A.M., pulse 120, respiration 36, temperature 102; P.M., pulse 130, respiration 40, temperature 106. 8th, A.M., pulse 120, respiration 40, temperature 103.5; P.M., pulse 136, respiration 48, temperature 106. 9th, A.M., pulse 160, respiration 40, temperature 104. Patient died February 9, at 12 M.

Autopsy twelve hours after death. Thorax: Heart normal, cavities contained no blood. Lungs: right lung did not collapse when the pleura was opened; lower lobe in second stage of pneumonic inflammation, left lung adherent to walls of chest by old pleuritic effusion. Lower lobe like that of right lung, congested and solidified and in second stage of pneumonic inflammation.

Abdominal cavity: liver, spleen and kidneys normal. Gall bladder distended with bile. Peyer's patches in ileum, prominent with ulcerations; solitary glands of small intestines and especially of ileum enlarged and ulcerated. White deposits in Peyer's gland and solitary glands.

In a case of yellow fever, which terminated fatally in the month of November, in the ward directly opposite to the one in which the preceding case was located, the temperature on the third and fourth day of the disease reached 104, while the pulse never exceeded during this period 100. This case (Martin H. King, a native of Ireland, age 26) terminated fatally on the fourth day; and the post-mortem examination revealed the characteristic lesions of yellow fever; fatty yellow heart, liver and kidneys; black vomit in stomach; mucous membrane of stomach highly congested.

Case of gastritis, fever and jaundice, resulting from exposure and bad diet. John Austin, age 16, laborer, admitted to Charity Hospital Nov. 30, 1876. Says that he had felt unwell for three or four days before admission. Complained of loss of appetite and diarrhea. Ordered light diet and bismuth mixture.

℞. Syrupi morphiæ . . . . ℥iij
Bismuthi subnitrates . . . . ʒiij
ℳ: Sig., teaspoonful three times a day or every four hours if necessary.

December 4, temperature of the axilla 101.4. December 5, 101; December 6, eyes and skin decidedly yellow, patient complains of feeling very heavy and depressed. Morning pulse 82, respiration 18, temperature 99.8; P.M., temperature 100.5. December 7, morning, jaundice increasing, decided yellow color of conjunctiva and skin; pulse 72, respiration 18, temperature 99.5; evening, pulse 72, respiration 18, temperature 100.5. December 8, urine high colored and loaded with bile, albumin absent; chlorids abundant; yellow color of conjunctiva and skin. A.M., pulse 74, respiration 18, temperature 99; evening, temperature 100.5. December 9, morning, pulse 80, respiration 18, temperature 95.5; evening, pulse 80, respiration 18, temperature 101.5. December 10, evening, pulse, 76, respiration 18, temperature 102.6. Urine loaded with bile; skin yellow, hot and dry. December 11, morning, pulse 80, respiration 18, temperature 99; evening, temperature 102. December 12, morning, pulse, 80, temperature 100.5; evening, temperature 101.6. December 13, during the past four days quinin has been freely given, the bowels having been previously evacuated by a mercurial purgative. The depth of the color of the skin is now decreasing. Morning, pulse, 84, temperature 99.8; evening, temperature 100.5. Continued to improve and was discharged.

We observe in this case in which jaundice accompanied fever, perhaps of a malarial character, that on January 10, with a temperature of 102.6, the pulse was only 76 beats per minute.

PERNICIOUS AND CONGESTIVE MALARIAL FEVER.

The rate of mortality in my surgical practice has varied with the season, the type of the disease and the manner of admission of patients. When from such causes as scarcity of funds, the lighter cases of disease were treated as out-door patients, and only those seriously ill admitted, the rate of mortality among the cases treated in the wards was relatively increased. At the same time the number of out-door patients prescribed daily in the wards was greatly augmented. I have carefully excluded from the statistics recorded in my "Medical and Surgical

Memoirs," those relating to out-door patients connected with the Charity Hospital, although during, some seasons they have exceeded in numbers those actually treated in the wards. During the twenty-seven months of hospital service, preceding April 1, 1872, the total number of cases admitted into my wards in the Charity Hospital was 1,111; of this number 856, or 77.9 per cent. were discharged, and the deaths amounted to 106, or 9.4 per cent. of the entire number treated; during this period, also, a large number of the cases which terminated fatally were brought in moribund. During the six months ending April 1, 1873, the total admissions numbered 213; of this number 145, or 68 per cent. were discharged. The deaths amounted to 39, or 18.5 per cent. During the first period the deaths amounted to 1 death in 10.4 cases; in the latter period, 1 death in 5.46 cases.

During a period of six months, Oct. 1, 1873 to April 1, 1874 the total admissions numbered 517; of which 436 were discharged and 51 died; the deaths amounting to 1 in 10.13 cases, or 98 per cent. During this period more than one-third or 19 deaths were caused by yellow fever, many of which were brought in moribund, with jaundice and black vomit.

During a period of twelve months, Oct. 1, 1874, to April 1, 1875; Oct. 1, 1875, to April 2, 1876, 765 cases were treated, with 65 deaths, or one death in 11.8 cases, or 8.5 per cent.

I have selected the following cases from the clinical records of my wards, to illustrate the condition in which many of the patients entered, and the effects upon the rate of mortality:

Case 1.—Pernicious malarial fever (malarial coma). Unknown man, age about 35, entered Charity Hospital Oct. 17, 1872; died in six hours. Patient brought to the hospital in an insensible condition, from which he could not be aroused; rapid feeble pulse; skin sallow with a decided tinge of yellow; extremities cold, congested and mottled, lips and fingers purple; passes urine and feces freely in bed; head and trunk hot; temperature of axilla 103. Sinapisms and alcoholic stimulants failed to arouse the patient; large doses of quinin administered by enema have no effect.

Post-mortem three hours after death: Blood vessels of brain congested with dark blood; lungs and heart healthy; mucous membrane of stomach and intestines slightly congested; liver slate-colored on the exterior and bronzed within; abundant pigment deposits in portal capillaries; spleen enlarged and softened; splenic mud contained numerous pigment particles; kidneys normal; gall bladder distended with dark green bile; cavities of heart contained fibrinous clots; blood of liver of a dark purple color.

Case 2.—Pernicious malarial fever (malarial coma). F. F. Payne, age 32, native of Virginia, entered Charity Hospital Oct. 28, 1872, died October 29, twenty hours after entrance. Has been exposed in swamps of Mississippi River. Admitted in a comatose state. Rapid pulse, congested purplish extremities. When aroused by violent shaking, appears to be in a state of great agitation and gives incoherent answers. Passes excrements in bed. Sinapisms, alcoholic stimulants and quinin produced no permanent effects.

Post-mortem six hours after death: Heart and lungs normal; fibrinous clots in cavity of heart; liver slate-colored on the exterior and bronze within; spleen enlarged and softened; gall-bladder distended with dark green bile. Stomach contained a considerable quantity of greenish-yellow fluid resembling bile; numerous pigment granules were observed in the structures of the liver and spleen.

Case 10.—Malarial congestion or pernicious fever. Charles Smith, native of Switzerland, age 60; entered Charity Hospital Oct. 18, 1872, and died October 19. During the summer and fall has been exposed to swamps and marshes. Entered in a state of wild delirium. Rapid feeble pulse; rapid respiration; prostration of muscular and nervous forces; capillaries of extremities congested; head and trunk hot; temperature of axilla 103, which continued up to the time of death. Passed feces and urine in bed. Sinapisms, diffusible stimulants and quinin produced only temporary improvement and the intellect was restored but for a short time. Intense jaundice appeared before death, which occurred in thirty-six hours.

Post-mortem six hours after death: Surface golden colored; heart and lungs normal; liver slate-colored on the exterior and bronze within; much pigment in portal and hepatic capillary network. Gall-bladder distended with dark almost black greenish bile; spleen enlarged and softened; kidneys healthy.

Case 3.—Malarial, congestive or pernicious fever. John Kerrin, age 39, native of Denmark. Has suffered for some time with malarial fever, in a low, unhealthy, marshy location. Entered Charity Hospital moribund, pulseless, comatose, Feb. 9, 1873; died in twenty-four hours, Feb. 10, 1873. Diffusible stimulants, sinapisms and sulphate of quinia induced only temporary improvement.

Post-mortem examination: Heart normal; contained fibrinous clots; malarial liver, slate-colored on exterior and bronze within, loaded with black pigment; spleen enlarged, softened and loaded with pigment.

Case 4.—J. Duber; malarial fever and dysentery; native of Germany, age 45. Has been exposed in swamps and has been subjected to scant diet and bad water. Entered Charity Hospital Nov. 12, 1872, and died November 19. Entered hospital in a most filthy and miserable condition; comatose; pupils contracted, pulse rapid and thread-like—so rapid and feëble that the number of beats are determined with difficulty. Bowels loose, passes feces and urine in bed. Has been sick for some time. Immense bed sores are forming over scapulars, sacrum and hips. Under the use of large doses of quinin and opium, carbonate of ammonia and alcoholic stimulants the patient rallied and at one time I entertained some feeble hope of his gradual improvement. The change, however, was only temporary, and death appeared to result chiefly from the profuse discharges from the bowels; patient died six days after entering the hospital.

Post-mortem six hours after death: Heart and lungs normal; liver slate-colored on exterior and bronze within with much black pigment; spleen enlarged and contained much pigment; ileum, colon and rectum much congested; numerous ulcerations in mucous membrane of colon and rectum. In the chronic dysentery of the Mississippi Valley and of the Southern States generally, I have found by post-mortem examination that ulcerations of the large bowels are almost always present.

Case 5.—Malarial congestive fever, with hemorrhage from the bowels. John Kelly, age 42, native of Ireland. Entered November 11 and died November 13. Cause of disease, exposure on banks of Mississippi and bad diet. Entered in a delirious feeble state with rapid feeble pulse; distended abdomen; enlarged liver and spleen; anemic, sallow, greenish-yellow malarial hue. Passes foul stinking feces in bed. On the night of the 12th the patient had a prolonged (congestive) chill, which was attended by a profuse discharge of fetid bloody matters in his bed. Quinin, opium, stimulants and sinapisms produced only temporary effects, and prolonged life only a few hours.

Case 6.—Malarial congestive fever and dysentery; profuse hemorrhage from bowels. F. Harrington, age 33, native of Ireland. Entered Charity Hospital Jan. 11, 1873, died January 21. Has been exposed to the prolonged action of malaria, cold, and wet, and bad diet in swamps along the Jackson R'y. Entered with feeble rapid pulse; sallow and anemic, jaundiced hue; liver and spleen enlarged; suffers with quotidian intermittent fever and painful chronic dysentery; discharges from bowels painful, frequent and loose. Under the free and continuous use of subnitrate of bismuth, opium and quinin, with milk and rice diet, there was marked improvement. On the night of January 18, patient had a prolonged and severe congestive chill attended with profuse hemorrhage from the bowels, which was followed by collapse and death.

Post-mortem six hours after death: Heart and lungs normal; liver slate-colored on exterior and bronze within, with much pigment matter; spleen enlarged; mucous membrane of large intestine ulcerated.

Case 7.—Pernicious fever and dysentery terminating fatally with profuse hemorrhage from bowels. Timothy Harrington, age 32, laborer; admitted to Charity Hospital Jan. 11, 1873. Patient states that he was taken two weeks ago with violent pain in the lower bowel, with frequent desire to go to stool, and great tormina and tenesmus. Bowels moved six or eight times in twenty-four hours. January 13, pulse 100, of good force and volume. Complexion anemic and greenish-yellow, spleen and liver enlarged and has been exposed to the action of malaria. Temperature 102 degrees. Applied blister on right iliac region for relief of pain. Administered 5 grains of quinin and 10 drops of the tincture of opium every three hours, until four doses were taken. Fever not affected by quinin perceptibly; patient restless and complains of pain in bowels. January 15, pulse 108, very feeble; patient feeble and weak. During the night of the 15th had a protracted and severe chill of the type ordinarily called congestive; extremities purple; pulse a mere thread. January 16, patient in a state similar to the cold stage of congestive fever; cold clammy sweat on the surface; complete prostration; pulse rapid and feeble; turpentine stupes and sinapisms were applied and quinin and alcoholic stimulants administered, both by the mouth and by the rectum. These measures brought on reaction and the patient appeared to be improving. The temperature of the axilla, however, during this period of depression and coldness of the extremities was above the normal standard, as will be seen by the following record: January 13, temperature 102, pulse 100, respiration 18. January 14, temperature 100, pulse 99, respiration 17. January 15, A.M., temperature 101, pulse 86, respiration 19. January 16, temperature 101.5, pulse 108, respiration 18. January 17, temperature 102, pulse 88, respiration 23. January 18, patient vomiting large quantities of biliary matter and utterly unable to retain anything on his stomach; A.M., temperature 101, pulse 84, respiration 18. January 19, during the nights of the 18th and 19th the patient was again affected with a prolonged congestive chill, attended with profuse hemorrhage from his bowels. His bed was literally flooded by the hemorrhage from the bowels. Pulse 128, respiration 18, temperature of axilla 100. I examined the urine with great care and interest in the case, as it bore striking resemblance to some cases of malarial hematuria. The urine contained no blood corpuscles and no albumin, but abundant urea, urates and uric acid; was of a bright straw color, strong acid reaction; specific gravity 1025. January 20, temperature 100.5, pulse 120, respiration 20. Died January 20.

Post-mortem. Twelve hours after death: Heart and lungs normal; liver enlarged, slate-colored on exterior, bronze within, loaded with pigment particles; spleen enlarged and softened; mucous membrane of intestine congested; large intestine ulcerated.

Case 8.—Intermittent fever arrested by full doses of the sulphate of quinia. James Davis, age 25, native of England, has been exposed to the action of malaria in the swamps of the Red River. Entered Charity Hospital January 19, discharged Jan. 27, 1873. Upon entrance complained of pain in right side. Had severe chill during night of 21st. Bowels opened with 10 grains of calomel, combined with 5 grains of quinin and followed with 5 grains of quinin every two hours, until 20 grains were taken. Under the action of the quinin there was a rapid subsidence of the temperature. The quinin was continued; about 10 grains being administered daily for several days; no return of chill and fever.

This patient had had chill and fever every alternate day up to entrance in hospital. The following observations illustrate the rapid fall of temperature under the action of quinin: January 21, 8 A.M., chill just passing off, temperature of axilla 106.3; quinin freely administered. At 8 P.M., temperature 102.3. On 22d, at 8 A.M., temperature 99.3; 8 P.M., temperature 99. On 23d, 8 A.M., 99.4; 8 P.M., 100. On 24th, 8 A.M., 98.5; 8 P.M., 98.4. On 25th, 8 A.M., 98.4; 8 P.M., 99.2. On 26th, 8 AM., 99.4.

Case 9.—Congestive fever; loss of muscular power, general paralysis, loss of memory. Recovery. Peter O'Brian, age 41, native of Ireland, laborer. Enjoyed good health up to November, 1872, when he left St. Louis, Mo., where he had been employed as a ditcher, with the intention of settling in New Orleans. On his passage down the Mississippi River to New Orleans he was robbed and left destitute. After arriving in this city he was for nearly a week without shelter or sufficient food, and slept at night on the levee without covering of any kind, during cold and inclement weather. This exposure and want of food so reduced the strength of the patient that he was compelled to apply for lodging at the police station, where he was "very ill with chills and fever." From the police station he was transferred to the workhouse, in which place "becoming much worse," he was sent to the Charity Hospital. He was brought in January 12, 1873, in an insensible comatose state, with loss of speech and power of motion. Lay insensible in the bed, passing his urine and feces involuntarily. Could not be aroused. At times the patient appeared to suffer with true malarial chills, attended with shivering, depression of the temperature of the extremities, elevation of that of the trunk and congestion of the peripheral capillaries, and followed by general diffusion of heat and high temperature.

Regarding the chills, fever and coma of malarial origin and aggravated by exposure and starvation, I placed the patient upon a solution of quinin:

℞. Quiniæ sulph.	gr. xxx.
Acidi citrici	ʒss.
Tinct. cinchonæ comp.	f ℥viij.

Mix: Tablespoonful in wine glassful of water, every four hours; and ordered beef tea and milk punch, every two hours.

Under this treatment, the improvement was slow but gradual, and on January 7, he was able to give a few coherent answers. The brain, however, was in such an enfeebled condition that he was able to give his name and answer a few questions, when his mind would wander and he would relapse into an unconscious condition. The quinin with tincture of cinchona and quassia, and the nutritious diet was continued, and on January 13, the thirty-second part of a grain of strychnia, in solution with the citrate of quinin and iron was administered three times a day.

℞. Strychnia sulph.	grs. ij.
Acidi citrici.	ʒij.
Quiniæ et ferri citrati	ʒiij.
Aquæ destillatæ	f ℥viij.

Mix: Sig., teaspoonful three times a day.

This patient was carefully observed by the students of the ward, and was also at regular intervals brought before the entire class in the amphitheater, in order that the gradual improvement of the mental and muscular power might be noted. The patient slowly progressed to complete recovery and was discharged at the end of sixty days.

CHRONIC MALARIAL POISONING.

Case 1.—Great anemia; fatal results. D. Hoffman, age 28, native of Germany, came to this country two years ago. After remaining in New Orleans one month, went to Franklin Parish to work on a plantation. Was taken sick with chills and fever three weeks before entrance into the hospital, and during this time had little or no medical attendance and had but indifferent supplies of food. Entered Charity Hospital, Oct. 28, 1873. Pale, anemic; hands bloodless and when held up to the light resembled semi-transparent light yellow and greenish wax. Countenance pale, yellowish green, swollen and bloated. Lips and tongue pale like the face; tongue pale, large, flabby, with the edges indented by the teeth. Action of heart loud, rapid and irritable, with loud anemic murmur. The anemia in this case was as intense as in the most pronounced cases of chronic malarial poisoning in the Mississippi valley. Belly and lower extremities anasarcous. October 29, 8 P.M., pulse 110, respiration 20, temperature 103.5. October 30, 8 A.M., pulse 104, respiration 20, temperature 101.5. October 31, 8 A.M., pulse 106, respiration 24 temperature 101.5; 8 P.M., pulse 106, respiration 24, temperature 101. November 1, pulse 100, respiration 20, temperature 101.5; 8 P.M., pulse 108, respiration 25, temperature 101.5. November 2, pulse 108, respiration 18, temperature 101.5; 8 P.M., pulse 100, respiration 20, temperature 100. November 3, pulse 116, respiration 28, temperature 101.6; 8 P.M. pulse 112, respiration 32, temperature 100,

November 4, pulse 104, respiration 22, temperature 101. The patient complained of pain in the epigastrium and region of heart together with great dyspnea. The difficulty of breathing appears to be clearly due to edema of the lungs, induced by the watery condition of the blood, and is similar to the universal anasarca with which all parts of the patient appeared to be afflicted. The distress in breathing and the pain in the heart increased during the night, and was attended with great thirst. The patient died at 8 A.M., November 5. Death apparently due to edema of the lungs. The preceding changes wrought in the blood and textures by the malarial poison were the chief causes of death.

Autopsy: Two and one-half hours after death. Body warm and limber in *rigor mortis*. Exterior, full bloated anasarcous appearance; cellular tissue infiltrated with yellow serum, color greenish yellow, resembles a Chinaman. Chest, lungs bloodless, pale, anemic; edematous. Heart enlarged, with dilatation of right auricle and ventricle; color of heart natural; when the textures of the heart were carefully examined under the microscope with magnifying powers of various degrees, the transverse striæ were found to be distinct and there were deposits of granular and pigmentary matter within and around the fibers of the heart as in yellow fever. Cavities of the heart distended with dark fluid blood. Specific gravity of blood 1023.3; specific gravity of serum 1017; solid matters in 1000 parts of blood 77.71; in 1000 parts of serum, 60.94; 1000 parts of blood in the patient contained not more than 17 parts of red blood corpuscles in 68 moist blood corpuscles, while in healthy blood the moist corpuscles may reach 600 parts; the liquor sanguinis 400 parts. In the present case the liquor sanguinis was 932 parts in 1,000. Under the microscope, the colorless corpuscles were found to be greatly increased, relatively to the colored corpuscles. Abdominal cavity: Liver slate on the exterior, and bronze within. Under the microscope the liver cells were found to be loaded with dark pigment particles, which were also deposited in large numbers within and around the portal capillaries. When sections of the liver were made with Valentine's double-bladed knife and examined under the microscope, numberless dark pigmentary masses were seen both in the portal and hepatic systems of capillaries. Liver firm, evidently undergoing a hardness or cirrhotic change which appeared to be due to the action of malaria. Gall bladder distended with dark green bile, about two fluid ounces. Bile thick and ropy. Specific gravity of bile 1022. Solid matter of bile 1000 parts, 82.19. The bile was actually of very nearly the same specific gravity as the blood, and contained more solid matter. Spleen enlarged and loaded with dark pigment particles. Kidneys firm and normal.

In the preceding case quinin, nutritious diet and alcoholic stimulants were used but without avail.

Case 2.—Chronic malarial poisoning; intense anemia; jaundice; high temperature; anasarca; fatal issue. P. Eghart, age 25, laborer, native of Germany. Has been in Louisiana eight months. Has been living in a low swampy region. Has had chills and fever, with little or no medication and with scant supplies of coarse food. Entered the Charity Hospital on the evening of Nov. 22, 1873. November 23, pale, sallow and greenish-yellow complexion, bloated, swollen features; extremities edematous; scrotum swollen; feeble prostrated condition; great prostration of nervous and muscular forces; rapid feeble pulse; loss of appetite; anemia complete; lips and tongue pale; when hands are held up to the light they present a wax-like almost semi-transparent appearance; tongue pale, flabby and indented at the sides; urine high colored but free from albumin and casts.

Before coming to this country this patient followed the trade of architect and enjoyed good health. His present deplorable condition is clearly referable to the action of the malaria of the swamps.

November 24, 8 A.M., pulse 112, respiration 21, temperature of axilla 105 degrees, 8 P.M., temperature 104.8. November 25, continues in the same condition only apparently more feeble; can take but little nourishment. Quinin administered freely; 8 A.M., pulse 116, temperature of axilla 103; 8 P.M., temperature of axilla 104 degrees. November 26, delirium, great prostration, jaundice quite intense. Pulse rapid and feeble; skin hot and dry; tongue dry and pointed. Great thirst, nausea and vomiting, Can retain no nourishment on stomach. It has been difficult to introduce nourishment by the rectum on account of the irritable condition of the bowels. Urine high colored but free from albumin and blood. Chlorids present but diminished in amount. 8 A.M., temperature 102; 8 P.M., temperature 103. Died at 6 A.M., November 27.

Autopsy three hours after death: Body warm, limbs flaccid. Exterior, color of surface yellowish-green; lower extremities edematous; belly full. Thorax: Pericardium contained about two fluid ounces of golden yellow serum. Heart, of the normal deep color of this organ in health; perhaps it would be more accurate to describe the color as of a deep purplish red; muscular structures of the heart firm. Under the microscope the muscular fibrillæ presented a normal appearance, with distinct transverse striæ; and there was also a complete absence of the yellow granular molecular matter and oil globules characteristic of yellow fever. Surely such cases as these now recorded, when the malarial poison has exerted its unobstructed action in healthy Europeans, must yield unexceptionable results in settling the character of the changes induced in the chemistry and minute anatomy of the organs by the malarial poison. Heart normal in size.

Microscopic and chemic examination of the blood from the cavities of the heart and from the vena cava:

Specific gravity of blood	1021
Specific gravity of serum	1015
Solid residue in 1,000 parts of blood	73.00
Solid residue in 1,000 parts of serum	58.00

The blood of this patient contained less than sixteen parts of dried colored blood corpuscles in 100 parts of blood. The true composition of the blood as compared with that of normal blood would be 64 parts of moist colored corpuscles and 936.00 parts of liquor sanguinis, against 600 parts of moist corpuscles and 400 parts of liquor sanguinis in healthy blood.

The blood from the cavities of the heart contained a large number of dark irregular masses of hematin of various sizes, from 1-2000 to 1-500 of an inch in diameter. These dark pigment masses were similar in all respects to those found in the textures of the liver and spleen. The blood also contained oil globules which accumulated upon the surface during the coagulation. Fibrin diminished, coagulum large and soft. Heart normal in size and structure. Lungs healthy with the exception of hypostatic congestion of dependent portions. Abdominal cavity: Stomach distended with gas, and pale; anemic in appearance. Contained a quantity of liquid deeply tinged by bile.

Liver: Enlarged and somewhat hardened; deep slate color on the exterior and bronze within. Liver cells contained but few oil globules, but on the contrary many dark pigment particles. Sections with Valentine's double-bladed knife, revealed under the microscope numerous irregular masses of dark pigment, deposited both in the portal and hepatic capillary network. The pigment was deposited on the walls of the veins and secreting structures as well as in the cellular interspaces. Gall bladder flaccid and contained about 50 grams of thin mucoid yellow bile, loaded with the desquamated cells of the mucous membrane of the gall bladder. Spleen, enlarged and softened, with numerous dark pigmentary particles. Kidneys, normal, with the exception of two slate-colored spots, about half an inch in diameter on each kidney. Discoloration due to deposits of black pigment particles.

Remittent fever. B. Casavan, age 27, laborer, native of France. Entered Charity Hospital Nov. 6, 1875. Patient entered with delirium and jaundice, and upon a superficial view resembled a case of yellow fever. November 7, 8 A.M., pulse 115, respiration 30, temperature of axilla 104.5. Tongue very red about the edges, furred in the center and cracked. Hemorrhage from both tongue and gums. Gums red and swollen. Upon inquiry I found that this patient had been working on the lower coast and had suffered with paroxysmal fever for about twenty days before entering the hospital. At 8 A.M., pulse 137, respiration 24, temperature 105.2. Notwithstanding the increase in the pulse, which differs from that of yellow fever, the appearance of this patient resembles closely that of yellow fever. A careful record of the pulse, respiration and temperature was kept and the urine examined daily; the case although presenting in its general features the appearance of yellow fever, was clearly not this disease, as shown by the following facts: 1, the temperature exhibited marked periodic depression and elevation as in paroxysmal fever; 2, the urine was wholly free from albumin and casts and manifested periodic changes as in malarial fever depositing during the remissions heavy deposits of urates and triple phosphates. The fever was finally and completely arrested by sulphate of quinia and the patient discharged December 1. The following is the record of the temperature, pulse and respiration:

Date.	Pulse.		Resp.		Temp.	
1873	M.	E.	M.	E.	M.	E.
Nov. 7	115	137	30	34	104.5°	105.2°
8	117	133	31	37	102	104
9	96	102	27	36	102	103
10	96	90	28	30	101.5	102
11	96	90	17	26	100	101.5
12	84	. . . .	30	. . . .	101.5	. . . .
13	90	96	30	30	102	101.2
14	102	96	30	24	103.5	103.5
15	80	90	18	30	100	102
16	80	80	21	21	101	103.5
17	90	. . . .	24	. . . .	102	. . . .
18	78	. . . .	21	. . . .	101	103
19	80	. . . .	18	. . . .	100.5	. . . .
20	75	. . . .	18	. . . .	101.5	102.2
21	82	72	24	22	100.2	100.6
22	82	. . . .	18	. . . .	100	101.2
23	84	. . . .	20	. . . .	99.5	102.1
24	84	. . . .	. . . .	. . . .	99	101
25	82	. . . .	18	. . . .	100.2	. . . .

PATHOLOGIC ANATOMY OF THE HEART IN YELLOW FEVER.

It appears to be well established that in malarial fever the pulse and temperature are subject to great and sudden variations, and that quinin has the effect of dimininishing the frequency of the pulse, so that in many grave cases of malarial fever, the pulse especially when the patient has been treated with quinin falls to a very slow beat as in yellow fever. This will be illustrated by the following statement of cases, which I have selected, as it were at random without any reference to a special selection:

Remittent fever. Henry Bouteau. Entered Charity Hospital Sept. 29, 1873; native of Germany. Had been working in Red River rapids, and had suffered with intermittent fever for two weeks before entering hospital. Pale, anemic, had

had chills every day. October 18 had a severe paroxysm of fever which is here recorded. Discharged cured October 27. Urine light color and without albumin. A.M., pulse 112, 80, 68; respiration 50, 47, 30; temperature 105.4, 100.5, 98. P.M., pulse 105, 85; respiration 47, 45; temperature 103.2, 101.

Remittent fever. I. Madden, age 22, native of Germany. Entered Charity Hospital October 1, with severe hemorrhage and high fever. Arrested temporarily by quinin. October 8, day of first observation recorded; headache, bowels constipated, tongue dry, furred, brown and rough like sandpaper. Quinin arrested the paroxysm and relieved the pulse and temperature. Urine contained bile but no albumin. A.M., pulse 94, 65, 67, 80; respiration 31, 22, 22, 20; temperature 100, 96.3, 98.7, 96.6. P.M., pulse 96, 75, 90, 64, 70; respiration 38, 28, 27, 23, 22; temperature 101, 103.6, 103.2, 98, 97.5.

Chronic malarial poisoning. L. Meyer. Entered Charity Hospital Oct. 3, 1873. Had been sick with chills and fever for two months before entrance into hospital. Entered in a state resembling congestive chill; delirious; rapid, feeble pulse. Disease arrested by quinin. Observations began October 8 and 9. Patient ordered quinin. A.M., pulse, 55, 50, 54; respiration 15, 15, 12; temperature 97.5, 97.8; 98.3. P.M., pulse 60, 61, 54, 53; respiration 18, 12, 15, 14; temperature 97.4, 98, 98.5, 99.

J. Kousben, age 30, native of Germany. Has resided in New Orleans three years. Occupation, brewer. Admitted Oct. 6, 1873. Sick for six days before entering hospital. Observations began on October 8. Entered with high fever, arrested by quinin. Tongue still furred; urine high colored but free from albumin. P.M., pulse 68, 72, 80, 78; respiration 18, 16, 15, 20; temperature 96.8, 98.5, 98.9, 99; P.M., pulse 76, 72, 72, 82, 74; respiration 18, 18, 16, 24, 20; temperature 97.4, 97.8, 99.4, 100.4, 94.

Remittent fever. R. Brethan, age 50, native of Germany, laborer. Resided in New Orleans three years. Had been sick for eight days with remittent fever. Entered with high fever; dry, rough, brown tongue. Urine red but free from albumin. Disease arrested by quinin, 20 grains every day. Recovered. A.M., pulse 74, 84, 66, 60, 68; respiration 18, 18, 17, 17; temperature 99.2, 100.2, 99.2, 99.6, 99.4; P.M., 76, 74, 68, 66, 70; respiration 20, 20, 22, 18, 18, 17; temperature 9872, 100.4 100.2, 100, 99.8, 99.7.

Remittent fever. Convalescence. G. Peifer, age 33, laborer, native of Germany. Sick fourteen days before entering hospital. Resided in New Orleans fifteen years. Admitted Oct. 7, 1873. Had fever and dry furred tongue at time of admission, with great prostration; urine free from albumin, Observations began on next day, October 8. Quinin admin, istered and with good effect. A.M., pulse 79, 74, 72, 68; respiration 15, 18, 16, 15; temperature 97, 98, 99.6, 98.8. P.M., pulse

96, 74, 72, 70, 66; respiration 18, 14, 15, 15, 14; temperature 98:3, 98.2, 98.5, 98.2, 99.1.

George Wilson, occupation wood chopper, age 20, native of New Orleans; attacked with dizziness, pain in head and fever Sept. 24, 1873. Entered Charity Hospital October 1. Complexion yellow, gums yellow, constipated, pain in epigastrium, brown furred tongue. Quinin arrested the fever. Observations began October 8, A.M., pulse 72, 71, 62; respiration 22, 23, 20; temperature 99.5, 97.5, 98.6. P.M., pulse 78, 67, 61, 66; respiration 22, 22, 22, 22; temperature 98.2, 98, 97.8, 98.5.

Remittent fever. F. McKenna, age 35, native of England, laborer. Attacked five days before entering hospital. Has resided in America one year; in New Orleans six weeks. Has been working on dredge boat. Admitted to Charity Hospital Oct. 13, 1873. On admission, high fever, brown, dry tongue. Observations began October 16, while patient was under the influence of quinin and he perspired freely. Quinin 15 to 20 grains daily. No albumin in urine. Discharged October 20. A.M., pulse 74, 62, 60, 57, 54; respiration 22, 14, 16, 18, 16; temperature 103.8, 99.4, 99.3, 97.25, 98. P.M., pulse 89, 60, 61, 56; respiration 22, 16, 18, 20; temperature 103.2, 90, 95.5, 99.

James Topliff, native of New York, age 30, clerk. Admitted Oct. 10, 1873. Resided in city one week. Disease remittent fever. Taken sick October 9. Urine light colored, no albumin. October 11, high fever, vomited green bile. Bowels opened with mercurials and fever treated with quinin, 15 to 30 grains daily. Observations began October 11. Discharged cured October 24. A.M., pulse 84, 72, 64, 88, 82, 60; respiration 14, 16, 16, 18, 20, 18; temperature 101.5, 100.7, 101, 103.1, 102, 98.4. P.M., pulse 80, 72, 72, 94, 78, 60; respiration 16, 18, 17, 24, 14, 20; temperature 101.8, 101, 101.4, 105.7, 99, 98.4.

Remittent fever. John Gilmore, age 24, admitted to Charity Hospital Sept. 30, 1873. Had been sick two weeks before admission to hospital. Native of Ireland; has resided in Louisiana three years. Urine free from albumin. October 13 had a paroxysm of fever, which was arrested by quinin. Discharged October 27. A.M., pulse 100; respiration 22; temperature 104.8. P.M., pulse 96; respiration 22; temperature 103.8.

Remittent fever. George Letz, age 40, German; resided in New Orleans five years. Entered Charity Hospital September 29. Sick four or five days before entering hospital. October 3 had a paroxysm of fever. Quinin arrested the fever and reduced the pulse. A.M., pulse 82; respiration 22; temperature 102.4. P.M., pulse 90; respiration 26; temperature 104.5.

Remittent fever. J. Dudley, age 24, native of New Orleans. Has had malarial fever for three consecutive seasons. Has been working on a plantation on the Bayou Têche. Entered October 24. Observations began October 31. Very obstinate case. Quinin finally arrested the disease. No albumin in urine. Improved on quinin and iron. A.M., pulse 86, 76, 78, 98, 92, 92, 82, 70, 80, 76, 84, 84, 83; respiration 20, 20, 18, 25, 20, 16, 20, 16, 20, 20, 16, 22, 23; temperature 102.3, 99, 100.6, 101.5, 102.7, 102, 102.7, 100.5, 101, 102.5, 101, 102. P.M., pulse 84, 92, 78, 98, 98, 86, 82, 84, 92, 88, 87, 86; respiration 20, 20, 18, 25, 20, 24, 20, 16, 16, 20, 19, 20; temperature 101.4, 101, 100.8, 101.5, 102.5, 102, 102.7, 101, 102.5, 100, 102.

Chas Schmidt, age 42, native of Austria. Has had yellow fever. Entered Charity Hospital and observations begun Oct. 23, 1873. No albumin in urine; delirious; tongue red at tip and edges, dark brown in the center. Pain in head, bones and epigastrium. Discharged October 31. A.M., pulse 120, 78, 88, 80, 64, 66, 58, 68; respiration 46, 24, 30, 27. 22, 20, 18, 28; temperature 105, 99.2, 104, 100, 98.5, 97, 98.4, 98. P.M., pulse 110, 98, 84, 66, 64, 52, 62; respiration 30, 35, 28, 25, 22, 24, 20; temperature 103.9, 103.5, 103.5, 99.1, 97.5, 97.8, 97, 99.1.

Chronic malarial poison. Remittent fever. Charles Tinbers. age 21, native of France. Has been in New Orleans six weeks; sick two months before entering. October ·2, had chill followed by fever. Severe headache, restless, pale and anemic. Urine red and free from albumin. Treated with full doses of quinin. Disease arrested November 5. Bile in urine; no albumin; specific gravity 1025. A.M., pulse 90, 88, 74, 72; respiration 22, 25, 18, 16; temperature 103.8, 100, 101, 101.2, 98, 98.2. P.M., pulse 108, 92, 90, 80; respiration 44, 26, 24, 19; temperature 100.5, 98.4, 100.8, 98.

Intermittent fever. John Gilmore, native of Ireland. Entered Oct. 30, 1893. Had chills and fever five days before entering hospital. October 31, when record of pulse and temperature was begun, had a chill at 100 degrees, followed by high fever, and at 7 P.M. the temperature was 106 degrees, pulse 108, respiration 24. Sulphate of quinia was freely administered and the fever ceased November 13 and did not return. This was a clear uncomplicated case of intermittent fever. A.M., pulse 72, 73, 74, 80; respiration 18, 20, 22, 18; temperature 99.5, 100.4. 100.8, 98.8. P.M., pulse 104, 79, 72, 74; respiration 24, 23, 18, 18; temperature 106, 101, 100.6, 98.

Intermittent fever. Michael Dolan: Sick one week before entering hospital, Oct. 23, 1873. Observations begun October 26; general appearance anemic, pain in epigastrium. Had fever on 23, when entering. Quinin freely administered, arrested the fever. A.M., pulse 60, 54, 48, 52, 52, 50, 60, 54, 58; respiration 28, 24, 26, 20, 24, 24, 19, 18, 19; temperature 98.5, 98, 98.5, 98, 98.8, 98.6, 97, 98, 98. P.M., pulse 52, 52, 46, 48, 52, 56, 55, 52; respiration 24, 22, 30, 20, 24, 24, 24, 18; temperature 96, 98, 98.2, 99, 99.5, 99.2, 100, 98.5.

In many severe and fatal cases of yellow fever the respiration is often greatly accelerated, as in the following case which entered Charity Hospital Oct. 10, 1873. Had been sick for five days. At 6 P.M., shortly after entrance, pulse 84, respiration 48, temperature 102.4. Patient delirious, restless and vomiting black vomit; suppression of urine. Died next morning at 8 o'clock.

CHAPTER VIII.

ALTERATIONS OF THE BLOOD IN YELLOW FEVER, INDUCED BY THE SUPPRESSION OF THE FUNCTION OF THE KIDNEYS AND LIVER, AND THE RETENTION IN THE BLOOD OF THE CONSTITUENTS OF THE URINE AND BILE.

The constitution of the blood is more or less altered in every case of yellow fever, but the changes are observed in greatest intensity in those cases in which there is partial or complete suppression of the action of the kidneys.

By careful experiments I have determined that during the active stage of febrile excitement in yellow fever, not less than 600 grains of urea are excreted by an adult during twenty-four hours. As the action of the kidneys is in many cases wholly arrested for various periods, ranging from one to four days before death, the whole amount of urea accumulating in the blood and remaining in the system may range from 600 to 2,400 grains. The gastro-intestinal mucous membrane, in such cases, eliminates the urea, both as urea and as carbonate of ammonia; and both these substances frequently enter into the constitution of black vomit, which in many cases is intensely alkalin from the presence of carbonate of ammonia, in sufficient quantities not only to neutralize the acid of the gastric juice, but also to give forth such strong fumes as to be visible as a dense white cloud, when a rod dipped in hydrochloric acid is held over the black vomit, freshly discharged from the stomach or removed from the viscus immediately after death.

The presence of ammonia in the fluids of the stomach in such cases is clearly not due to subsequent changes, but is referable to the decomposition of the urea in the stomach, after the manner which has been pointed out by Frerichs, Bernard and other observers. In such cases the blood in the cavities of the heart and in the large vessels is black, and does not form a clot, and the solvent properties of the ammonia are manifest not only in the dissolution of the fibrin, but also in the altered appearance of the colored blood corpuscles, and in the rapidity with which they disappear when the blood is allowed to stand.

One of the most striking facts which arrests the attention of the observer in post-mortem examinations in this disease, is the apparent abundance of the blood in the various textures, its fluidity, and also the bright arterial hue which it rapidly assumes upon exposure to the atmosphere. This condition is referable to the distension of the blood vessel system, in consequence of the failure of the kidneys to eliminate the watery elements, as well as to the action upon the blood of the excrementitious materials. While it is true that urea is present in healthy blood in small amount, as may be determined not only directly by chemic means, but also by a simple calculation, as has been done by Dr. Goodfellow ("Lectures on the Diseases of the Kidney, generally known as Bright's Disease and Dropsy," London, 1861, pp. 67–69), I have at the same time, by careful analysis, shown that the urea is greatly increased in the blood of yellow fever, amounting in some cases to near 2 per cent. of the whole mass of blood.

The following is the calculation of Dr. Goodfellow, with reference to the amount of urea which must necessarily be present in healthy blood:

No physiologist denies that the urea is merely separated from the blood. It is universally acknowledged that the kidneys have no converting power. It can not be supposed, therefore, that all the urea contained in the blood goes directly to the kidneys; and consequently it follows, that only that quantity which passes through these organs is freed from this excrement at every successive revolution of the circulation. The following calculation will show this clearly: We will take the case of a healthy man, excreting every twenty-four hours about 360 grains of urea. This quantity divided by 24 gives 15 grains every hour, and still further reduced, one-fourth of a grain every minute. We will suppose the quantity of blood in his body to be thirty pounds, and that it takes from a minute to a minute and a half, to complete the circulation. We will suppose, moreover, that the kidneys constantly contain about three ounces of blood, and that about five ounces pass through these organs every minute. As we get, then, a quarter of a grain of urea from the five ounces passing through the kidneys, in this space of time, it is reasonable to infer that there will be left in the remaining mass of blood (475 ounces), $23\frac{3}{4}$ grains of urea.

The quantities which Dr. Goodfellow has conjecturally assumed are, in the one case too small, and in the other too large, and he has subsequently added the following correction of the preceding calculation:

In one of the elaborate tables, in Dr. Parkes' work on the urine, it appears that the mean amount of urea secreted by males in twenty-four hours, as furnished by 250 analyses, made by twenty-four eminent chemists, is 512.4 grains, varying from 286.1 grains, the lowest, to 688.4 grains, the highest; and in females, from 260 to 400. The amount of blood (thirty pounds) is more than the area of the heart and blood vessels in a man of average size can accommodate, notwithstanding that it is the quantity as calculated by eminent physiologists. The amount of blood expelled from the left ventricle at each contraction, as supposed by Valentin (five ounces), and Volkmann (six ounces), is probably too large. The calculation is made on the supposition that at each contraction of the left ventricle from two and one-half to three ounces of blood are expelled, and that of the mass of blood so thrown into the aorta, from half a drachm to a drachm is propelled by the kidney, and that the same quantity makes its exit simultaneously by the vein. Supposing, then, that there are from seventy to seventy-five ventricular contractions in a minute, the total quantity passing through the kidney during this space of time, will therefore amount to from five to eight ounces. The calculation does not require the demonstration of the exact quantity which is always in the blood, but that there necessarily must be some urea constantly in it, and more than is generally supposed. The excretion of urea is always going on more or less quickly, and also the formation of this substance from the waste of the protein tissues, and from the same principles in the food, in greater or less quantity according to the state of the body, as to exercise or food; and as its transmission into the blood through its principal conduit, the thoracic duct (M. Wurst, as quoted by Bernard), is also constant, it follows that there must be more or less of urea in the blood at all times.

With respect also to the length of time required to complete the circulation, some facts of importance have been omitted in the calculation. Now that we have more correct notions with respect to secretion, most physiologists will show that although a complete revolution of the circulation may be effected in from a minute to a minute and a half, as deduced by calculation (Valentin and Volkmann), and by experiments (Poiseuille, Bernard and Blake), the blood really moves with varying velocity in different secreting and excreting organs; influenced as the circulation is probably by the peculiar affinities, and the diameter and arrangements of the capillaries and other minute vessels. We observe that every secreting organ has some peculiarity in the arrangement and size of the vessels, and in the thickness of their walls. This arrangement must affect to some extent the velocity of the circulation, and affect also the character of the blood plasma transuded into the tissue, from which the secreting agents are to separate the peculiar constituents of the secretion. Taking the double circulation of the blood in the kidneys into consideration, then, it is probable that the blood does not pass through them so quickly as through some other organs, and that the whole mass of blood does not pass through the heart every minute or minute and a half, although a quantity equal to it may do so; some portions may, in fact, pass twice or oftener through the heart in this space of time.

On the assumption that there are twenty pounds of blood in a man's body, and that one ounce and a half of urea is excreted in twenty-four hours, Marchand calculated that the blood contained the 15-360 part of its weight of urea.*

The results of these investigations on the condition of the blood, and upon the chemic constitution of black vomit in yellow fever, sustain the view held by MM. Prevost and Dumas, Claude Bernard, Ricord, and other physiologists, that urea after having been generated in the blood and tissues, is simply excreted by the kidneys, and does not confirm the doctrine taught by M. Herman, Dr. Beale and others, that urea and uric acid are not only eliminated but are formed by the renal tissue. And careful physiologic experiments which I have performed have been confirmed by the results of the inquiries of M. Gréhaut (*Archives de Physiologie Normale et Pathologique*, Sep et Novembre, 1870—*American Journal of Medical Sciences*, October, 1871, p. 530), which extended over several years. According to this careful observer, immediately after nephrotomy in the fasting dog, urea begins to accumulate in the blood, its increase being manifest within three hours after the operation; the increase of weight of the urea in the blood and in the lymph, twenty-four hours after the ablation of the kidneys, is equal to the weight of it that would have been excreted by the healthy fasting animal in the same space of time; the accumulation of urea in the blood in the hours that succeed nephrotomy, follows the same march as after ligature of the ureter. After ligature of the ureter, the quantity of blood circulating through the kidney of the side diminishes; under normal conditions the blood of the renal vein always contains less urea than the corresponding artery; in an animal in which the ureters have been ligatured, the renal venous blood obtained twenty-four hours after the operation contains as much urea as the renal arterial blood, so that the tissue of the kidney neither excretes nor secretes any more.

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* Lectures on the Diseases of the Kidney, generally known as Bright's Disease and Dropsy, by S. F. Goodfellow, M.D., Ch.C.. London, 1861, pp. 67-69.

Finally, M. Gréhaut has shown, that ligature of the ureter and nephrotomy are two operations that are identical in their results; they both suppress the eliminative function of the kidneys, while they form no obstacle to the formation of urea, which takes place outside of the kidneys.

Whatever theory may be held as to the cause of the peculiar phenomena, denominated uremia, whether that of the poisoning of the blood by the urinary constituents generally, or by urea especially, or by the product of its decomposition, viz., carbonate of ammonia, or by the edema of the brain from the accumulation of the watery constituents of the blood, attended by serous apoplexy and irritation of the brain and its membranes; it is evident that in many cases of yellow fever, the fatal issue is determined chiefly by the retention in the blood of the constituents of the urine. To this cause must be attributed to a great extent, the restlessness, nervous agitation, intoxication, delirium, convulsions and coma characterizing the stage of calm or depression in many cases of yellow fever.

NOTE.—Dr. Carl R. Braun, in his valuable work on the "Uremic Convulsions of Pregnancy, Parturition and Childbirth," has given the following summary of the state of our knowledge at the time of his work, which we here reproduce from the important bearing which it has upon similar phenomena in yellow fever:

"Eclampsia parturientium is commonly the result of uremic intoxications arising from Bright's disease of the kidneys, and produced mostly by carbonate of ammonia in the blood, perhaps also by extractive matters in the urine.

"This appears from the following analysis:

"*a*. All observers at present agree that urea retained in the blood is not, as such, the cause of the uremic symptoms.

"*b*. Lehman (Physiol. Chemic., Vol. II, s. 245) and Frerichs almost simultaneously and independently of one another arrived at the conviction that cause of the uremic phenomena is to be sought for in the ammoniacal contents of the blood, produced by the transformation of urea into carbonate of ammonia.

"*c*. The investigations of Frerichs, Litzmann, the author (Dr. Carl R. Braun), Heller, Kletznsky, Ofpolzer, Gegenbauer, and others have demonstrated that in the eclamptic, urea and carbonate of ammonia developed by its decomposition are generally found in considerable quantities in the fresh blood, that from the presence of these materials in the blood the occurrence of uremic eclampsia may be prognosticated, and that these substances are observed also in the blood of children born of uremic mothers.

"*d*. Chemic analysis, however, can not always, even during the most violent eclampsia, discern the presence of carbonate of ammonia in fresh blood, as is shown in an observation made on August 4, 1854, by Gustav, Braun and Heller and communicated to me. The blood, drawn from a vein after the sixteenth eclamptic fit, separated itself into clear serum and a light red, bulky, moderately consistent clot, covered with spongy fibrin, tinged yellow by a gall pigment. The strongly alkalin serum had a specific gravity of 1025, and contained much casein and bilephæin. The serum filtered after being treated with alcohol, contained little urea and sugar, much cholesterin and choliac acid. The blood then had not the uremic, but the cholemic constitution, as it contained no carbonate of ammonia, but all the elements of the bile. This observation, although it does not stand alone, can not be used to invalidate the theory of the very frequent coincidence of eclampsia and uremia. It rather points out that the excrementitial elements of bile may produce cholemia in the living body, and be likewise a cause of eclampsia.

"*e*. According to the very careful experiments of Mettenheimer (Mettenheimer, C., Archiv. f. Voissenschriftliche Heilk. I. 4,) Beneke, and Reuling, (Reuling, W., Inaug. Diss., Gussin, 1854,) all healthy and sick individuals have the power of expelling ammonia under certain circumstances. In the lungs of the healthy, ammonia is in general absorbed rather than expelled; hence the vapors produced in holding before the mouth a glass rod wet with dilute muriatic acid, are never capable of indicating the degree of uremia in Bright's disease.

"*f*. Normal blood when fresh drawn does not contain ammonia, as Reuling has proved by a very simple but very delicate qualitative method with logwood paper. In several diseases, as caries of the teeth, angina tonsillaris, typhus, pyemia, ischemia, and blenorrhea of the urinary bladder, we sometimes find in the blood carbonate of ammonia, just as in uremia and Bright's disease. Hence carbonate of ammonia in the blood can not be regarded as a characteristic indication of uremia, and in many constitutions uremia may be produced by extractive matters in the blood.

"*g*. After weighing the objections raised against the theory of the intoxication of the blood by carbonate of ammonia, Litzmann has arrived at the following conclusions: the fresh blood of healthy individuals never contains ammonia. In the majority of the cases of uremia, the blood does contain ammonia, which has been formed by the decomposition of urea formed in the blood and retained in it, or by the decomposition of the urea that has been secreted in the urinary passages and has returned into the circulation by absorption. The presence of ammonia in the blood is indicated by the increased ammoniacal contents of the expired air; but this increase can not of itself be considered a proof, for it may be produced by the admixture of ammonia formed in the cavity of the mouth by decomposition of the oral secretions and of remains of food in the case of carious teeth, etc. In the vomited fluids and the contents of the intestine in cases of uremia, carbonate of ammonia is not unfrequently discovered. Sometimes there is an alkalin reaction of the sweat (Litzmann, Rühle). The urine may contain ammonia, even when it has an acid reaction (Brüke). But, on the other hand, presence of ammonia in the blood is not by any means a sign of uremia exclusively, for it has been exceptionally observed (Reuling) in other diseased conditions (typhus and pyemia) when the urinary secretion was not disturbed. On the other hand, cases undoubtedly occur where notwithstanding obstructed secretion of urea by the kidneys, and the occurrence of all the characteristic symptoms of uremia, the blood does not contain any ammonia and the ammoniacal contents of the exhaled air are not increased (Reuling), but when, on the contrary, undecomposed urea is found in the transudations from the blood, and in the sweat especially, may be in such quantity as to be left on the skin in the form of white crystalline dust. (Schottin, Archiv. f. Physiol. Heilkunde, Vol. x, xi, xii; Fiedler, De Secretion Urinæ per Cutum, Dissit. Inaug, Med., Leipsic, 1854). The cause of uremic phenomena can not therefore be sought for in the decomposition only of the urea retained in the blood into carbonate of ammonia.

"*h*. Whether the accumulation of extractive matters in the blood, in consequence of suppression of the urinary secretion is the cause of eclampsia (Schottin, Reuling) or not, is a question for the future to decide. Hoppe found, in a case of a uremic poison, besides urea, three times the usual quantity of extractive matters in the serum of the blood, and also the muscles saturated with their excretory products. The quantity of kreatin obtained from them was five times as much as the normal amount.

"*i.* Wieger, on these grounds regards uremia not so loosely as the ancients, who held it to be a metastasis of the urine, and not so exclusively as Frerichs, who considers it an intoxication by carbonate of ammonia, but as a consequence of Brightian exudation into the kidneys which in its chemic relations is characterized by retention of water and excrementitial matters in the blood, which itself, from the loss of blood corpuscles and albumin is impoverished in these elements. But it is uncertain whether the chief part in the combined operation is to be ascribed to the excess of serum or to the diminution of the albumin and blood corpuscles. Hydremia, however, is never the cause of the nervous symptoms, but has only a predisposing action.

"*j.* From the results of chemic analyses it is certain that in uremia, carbonate of ammonia and urea are often found in the fresh blood and are rarely wanting. Although the essential nature of uremia is always coming into clearer light in consequence of the rapid strides of organic chemistry, yet we know enough already to assure us that eclampsia parturientium is always caused by the presence in the blood of an excess of excrementitial matters and also generally by uremic intoxication."

To the action of the same cause also may certain changes of the blood be referred, as the dissolution of the fibrin and the rapid changes which the colored blood corpuscles undergo after death and after removal from the living body. During the healthy action of the kidney, as has been shown by Simon, of Berlin, and Bernard, of Paris, fibrin disappears from the blood passing through this organ and is most probably converted into albumin, and the increase of the fibrin in Bright's disease has been referred to the cessation of this change of the fibrin in the diseased kidney; but in yellow fever, notwithstanding that the function of the kidneys may be wholly arrested, there is almost complete disappearance of the fibrin of the blood, so profound is the action of the defibrinating cause. That very important changes take place in the blood during the healthy action of the kidneys, from the separation of the urea and other constituents of the urine, is evident from the fact observed by Bernard, that when the kidney is active, the blood issuing from the renal vein has a bright sparkling red color, but if the secretion is more or less diminished or altogether suppressed, not only is the blood issuing by the vein dark colored, but the whole organ assumes a purplish tint, more or less deep. This change of color has, with some show of reason, been referred chiefly to the non-separation of water during the suppression of the function of the kidney.

Nasse has shown that if blood be diluted with water it assumes a dark red color; if the blood be previously dark colored, it becomes still darker on the addition of water; and if the blood corpuscles be examined under the microscope, they are found to be distended, to have lost their discoid form, and to have become spherical; the blood collectively therefore must appear darker, since each individual corpuscle has become converted into a spherical mirror, from which the red rays are scattered and reflected.

It has been shown by the experiments of several physiologists, that when the red blood corpuscles are in normal amount, a certain quantity of urea added to the blood scarcely produces any effect upon them; but on adding the same quantity to the same amount of blood, from which a considerable number of blood corpuscles had been withdrawn, a very striking effect was at once observed—the corpuscles rapidly assumed a vermilion tint, and sank to the bottom, presenting the appearance to the naked eye of fine vermilion; and on examining this sediment with the microscope it was found to consist entirely of red corpuscles, reduced to about a sixth of their natural size. The effect of the urea seemed to be in exact relation to the quantity of red corpuscles in the dependent portions of the blood, resembling fine vermilion, as in the preceding experiments, and this appearance must not only be referred to the loss of fibrin, but also to the action of the urea.

The blood of yellow fever containing more or less urinous excrement, and with a physical alteration and actual diminution of both the albumin and fibrin, is not only ill adapted to the nutrition of the organs, and actually conveys to the stomach materials which when eliminated alter so completely the properties of the gastric juice, as not only to arrest digestion, but also to irritate and corrode the mucous membrane; but it is also by its physical and chemical constitution unsuited to the maintenance of the general capillary circulation.

Both Bernard and Poiseuille have observed that fibrin when in intimate mixture with a due normal proportion of albumin, facilitates the movement of the blood; but if the fibrin be withdrawn the blood globules fall to the most dependent part, and obstruct the capillary circulation.

Bernard has recorded the observation that when the capillary circulation in the web of the frog's foot is examined under the microscope, the globules are seen suspended nearly uniformly in the liquor sanguinus; but if we examine this circulation in an animal whose blood has been defibrinated, we see the globules fall to the most dependent part, while at the superior parts, pure liquor sanguinis alone circulates. If a horizontal vascular trunk bifurcates in two divisions, not situated on the same horizontal plane, the lower branch will be plugged by the accumulation of the globules, while the upper branch will be full of serum.

Not only are similar causes active in producing the capillary congestions which are so characteristic of yellow fever, but the altered albumin and fibrin coagulate in the secreting and excreting structures of the liver and kidneys after its transudation through the walls of the capillaries and biliary and urinary tubes. To these changes of the blood, therefore, must be referred, to a great extent, the suppression of the functions of the liver and kidneys; for it is very evident that in the case of the liver the obstruction does not exist in the biliary or hepatic ducts, but in the biliary tubes.

It is evident that certain changes in the blood, as well as certain nervous symptoms in yellow fever, are due to the retention of bile in the blood.

Next to black vomit, jaundice occurring in the febrile stage of yellow fever is the most fatal symptom. Many observers regard jaundice as unfavorable in proportion to the earliness of its development; some writers considering jaundice as surely indicative of a fatal termination, when appearing upon the first or second day, or up to the third, fourth or fifth day.

Mr. Maher says that the period at which jaundice makes its appearance during the course of the disease is, according to the observations of Dr. Belot, a prognostic sign of great certainty. When it appears toward the third or fourth day, death will infallibly ensue; if it occurs toward the fifth or seventh day, the probability of a fatal issue is greater than that of recovery; finally, when it manifests itself only after the seventh day from the period of attack, recovery is almost certain (*Rapport Médicale sur l'Epidémic de Fiévre, Jaune*). The most valuable statistics with reference to this symptom have been furnished by Daniel Blair (Some Account of the Last Yellow Fever Epidemic of British Guiana." Third Edition, London, 1852, pp. 79-83). According to this accurate observer: "Yellow skin was always a sign of great intensity of disease. Among the 2,071 milder and graver cases, 385 had *yellow skin;* and of these 385, 178 died. Thus the proportion of cases in which the symptom appeared was 18.54 per cent., and the rate of mortality of the symptom was 46.23 per cent.

The following table will show the number of cases in which yellow skin was observed on different days of the disease, the number of deaths, and rate of mortality for each day, and rate per cent. of symptoms:

YELLOW SKIN AND BLACK VOMIT.

122 Deaths.						22 Recoveries.					
40 Cases Simultaneous.		45 Cases preceded.		22 Cases Succeeded		6 Cases Simultaneous.		6 Cases preceded.		10 Cases Succeeded	
Day of Disease.	No. of Cases.	No. of days preceded	No. of Cases.	No. of days succe'd'd	No. of Cases.	Day of Disease.	No. of Cases.	No. of days preceded	No. of Cases.	No. of days.	No. of Cases.
3d.. .	3	1	28	1	25	3	1	1	3	1	5
4th . .	14	2	10	2	4	4	1	2	2	2	2
5th . .	9	3	5	3	2	5	2	4	1	3	2
6th . .	10	4	1	7	1	6	2	. . .	. . .	4	1
7th . .	3	5	1	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .
8th . .	1	0	0	. . .	. . .	. . .	. . .	. . .	. . .	. . .	. . .

Black vomit, although a very unfavorable symptom, and more so than yellow skin, being as 75.68 to 46.23, is still not necessarily fatal. Out of the 366 cases of it, 277 only died, giving the centesimal mortality just stated, viz., 75.68. Yellow skin and black vomit were closely associated as to the time of their appearance, but the former was generally the antecedent (fortunate for the patient when otherwise). Thus in 139 ascertained cases, the former preceded the latter in 51 instances, appeared simultaneously in 46, and succeeded it in 42 instances. The double symptom of yellow skin and black vomit was of all others the most dangerous, and especially when the latter succeeded the former, and within twenty-four hours. The co-existence of these symptoms was observed in 144 cases, being 6.95 per cent. of the 2,071 mitior and gravior cases admitted to the Seaman's Hospital. Out of these 144 cases, 122 died, making the mortality of the conjoined symptoms 84.72 per cent.

The following table shows the day of the disease on which yellow skin and black vomit occurred simultaneously, also the number of cases in which the former preceded or succeeded the latter, and the number of days it so preceded or succeeded:

TABLE SHOWING THE NUMBER OF CASES IN WHICH YELLOW SKIN WAS OBSERVED ON DIFFERENT DAY OF YELLOW FEVER, THE NUMBER OF DEATHS, AND RATE OF MORTALITY FOR EACH DAY.

Day of Disease.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Unascertained.
Total number of cases.	8	16	46	89	86	66	30	18	7	8	5	.	.	3	1	2
Number of deaths	1	4	18	44	53	27	13	10	1	3	2	.	.	. . .	1	1
Rate of Mortality	12.5	25.0	39.01	49.45	61.62	40.90	43.33	55.55	14.28	37.5	40.	.	.	. . .	.	. .
Rate per cent. of Symptoms. .	0.38	0.76	2.22	4.20	4.15	3.14	1.45	0.87	0.34	0.38	0.29	.	.	0.14	.	. .

I have confirmed, by careful chemic analysis of the blood, and of the nervous structure and organs, the view held by some pathologists, that the yellow hue in jaundice observed in many grave cases of yellow fever is due to the presence of bile in the blood. By careful post-mortem examinations, as well as by attentive observation of the amount and chemic constitution of the urine in this disease, I have been able to refer the accumulation of the bile in the blood in this disease to two causes, viz., structural alterations of the liver, attended with desquamation of the excretory cells, obstruction of the biliary ducts, with altered fibrin and albumin and oil, and the accumulation of free oil in the excretory cells, and structural alterations of the kidney, resulting in impairment if not total suppression of its function.

Bile is a very common ingredent of the urine in yellow fever, even in those cases which terminate favorably, and as long as the functions of the kidneys are fully and freely performed, the blood may be relieved from any great accumulation of bile, even when the lesions of the liver are well marked, and from this cause the urine may be loaded with bile. When the kidneys cease acting, the bile as well as the urinary constituents rapidly accumulate in the blood, and certain changes in the physical and chemical characters of this fluid are referable to the bile, as well as to the urinary constituents.

In order to ascertain the effects upon the various functions resulting from the absorption of a large quantity of bile into the blood, but particularly with the object of tracing what becomes of the bile in the blood—whether it is transformed, or is excreted as bile, and, if transformed, what may be the nature of the changes it undergoes—Frerichs performed a series of experiments, the general results of which were as follows: some of the animals died under symptoms of violent dyspnea (obstruction of the capillaries of the lungs), owing to the entrance of air into the lungs, or to the too great consistency of the injected fluid; twenty-nine of the experiments, however, succeeded in such a way that their results could be made use of. In no case did any remarkable derangements of the nervous functions follow the injection; in no case was stupor, convulsions or retardation of the pulse observable; vomiting, however, occurred repeatedly in about one-fourth of the experiments, and in some of these cases also the injection of the bile was followed by some drowsiness. The character of the urine which was voided after the injection varied; sometimes it contained a larger or smaller quantity of coloring matter, and at other times it contained none; the former was the case in nineteen, and the latter in ten, of the twenty-nine experiments. The urine containing coloring matter was always passed in small quantity; it was of a greenish-brown color, became turbid upon cooling, and then appeared green, rapidly depositing flakes which, under the microscope presented a finely granular appearance. When collected upon a filter, this deposit formed a dark grass-green layer, which dried readily, and which, when recently dried, exhibited the characteristic properties of the coloring matter of bile, becoming decomposed by the action of impure nitric acid, or by a mixture of sulphuric and nitric acids, with a lively play of colors of green, blue, violet and red. The reaction of the urine was, in most cases, neutral or alkalin; the removal of the flakes of coloring matter was speedily followed by an abundant deposit of triple phosphates; the specific gravity of the urine varied. When the kidney is examined after death, in these cases, the tubes are deeply tinged by the bile contained in their secreting cells, and some tubes are nearly or quite filled with cells which have been thrown off, while others have been formed upon the basement membrane beneath them.

Dr. Johnston concludes from these facts, that when the blood circulating through the kidneys contains an excess of bile, the renal cells, in striving to eliminate these materials, become deeply tinged by it, and many of them are so far modified as to be shed by a process of desquamation.

The bile exerts a direct effect upon the kidneys, without doubt, inducing congestion and desquamation of the excretory cells in yellow fever, but the albuminuria, hematuria, and even total suppression of the functions of these organs, can not be referred wholly to the direct irritant action of the biliary products, but must, as in the albuminuria and desquamative nephritis of scarlatina, be referred to other causes also, as the irritant action of the poisons exciting the diseases, and the morbid condition of the blood induced by the action of specific poisons, as well as by the altered bile. That the retention of bile in the blood is capable of inducing profound alterations in its constitution, is evident from the fact that in all cases of jaundice which last for a length of time, the blood becomes impoverished by a diminution in the proportion of red globules and fibrin, and a tendency is developed to hemorrhages from the various mucous membranes. While this tendency to hemorrhage is practically observed in conjunction with cerebral symptoms and other indications of blood poisoning in cases of jaundice, when there is no obstruction to the bile ducts, it also occurs in cases of mechanical jaundice of long standing, in which the immediate cause of death is not unfrequently copious hemorrhage from the stomach and bowels.

While Frerichs observed in his experiments no marked effects upon the action of the heart or nervous system, it is well established by clinical observation that the presence of bile in the blood frequently retards the action of the heart and diminishes arterial tension, the pulse becoming irregular and falling to 50, 40, or even 20, and presenting the same characters as the pulse in yellow fever; and it is equally well established that acute delirium, stupor, coma, muscular tremors and convulsions may supervene in cases of jaundice, where there is no obstruction of the ducts, as well as in cases of long standing mechanical obstruction. The fact that slowness of the pulse is not an invariable symptom of jaundice has been explained by supposing that it is due to one particular ingredient of the bile, which does not exist in the blood in all cases of jaundice. Thus, Röhring has shown by experiments upon animals that the biliary acid salts exercise a specific paralyzing action upon the heart and retard its action, while bile pigment has no such effect; slowness of the pulse, therefore, in jaundice has been referred to the presence in the blood of unchanged biliary acids. Notwithstanding that the blood of the human subject may be saturated with bile in jaundice, for long periods of time, without the manifestation of any serious cerebral symptoms, at the same time there are certain cases of suppression of the function of the liver, attended with cerebral symptoms of the most violent character, hemorrhage from the bowels, and profound alterations in the blood and urine, which can only be referred to the presence in the blood of the altered secretion of the liver.

The theory advanced by Dr. Austin Flint, that the cerebral symptoms in jaundice are due to the retention of cholesterine in the blood, does not solve the question; for if the retention of all the elements of the bile in the blood does not give rise to cerebral symptoms, it is impossible to understand how the symptoms can result from the retention of cholesterine alone, which is one of the constituents of bile.

Cholemic eclampsia, attended with jaundice, and rapidly fatal tonic and clonic convulsions and coma, as well as that peculiar form of jaundice which is characterized by vomiting of blood, bloody stools, convulsions, coma and a rapid progress and fatal issue, evidently arise from the alterations induced in the blood by the constituents of the bile, and the products of their decomposition, resulting from acute atrophy of the liver, and present, not only in some of the symptoms, but also in the acute fatty degeneration of the liver. and infiltration of the organ with oil, and the alterations of the heart and kidneys, a striking analogy to the grave cases of yellow fever.

It is still a matter of dispute and doubt which element of the bile, or what products of the decomposition exercise the baleful influence on the nervous system in acute yellow atrophy of the liver. According to Frerichs, the formation of leucine and tyrosine, crystalline products of the decomposition of albuminous substances, may produce the nervous disturbances. Virchow, however, feels himself unable to grant this, because leucine and tyrosine are also found in typhus and exanthematous diseases, and may possibly be formed not until after death. Bamberger, on the other hand, thinks it more probable that they are produced by the acids of bile, and by the possible products of their decomposition; and Dr. Budd contends that they are due to some peculiar noxious matter evolved *in consequence of decomposition*, in the lobular substance of the liver; while Dr. Charles Murchison holds that the poison producing the cerebral symptoms is more probably generated in the blood and throughout the body generally than in the liver particularly.

The investigations of physiologists have shown that the liver not only excretes bile, but it forms grape sugar, and elaborates certain constituents of the blood, and exercises a continuous and important influence on the metamorphosis of matter constantly taking place in the blood and tissues.

That the arrest of the function of the liver checks or modifies the normal healthy metamorphoses of the blood and tissues is manifest, not only in the physical alterations of the blood in acute atrophy of the organ, but also in the great diminution of urea, and the appearance in large quantities of leucine and tyrosine, which possess a composition intermediate between it and the albuminoid components from which urea and uric acid are derived.

In acute atrophy of the liver, in acute phosphorous poisoning and in yellow fever, diseased states which have many symptoms and pathologic lesions in common, it appears that the albuminoid substances of the blood and organs, are split up into nitrogenous and non-nitrogenous combinations; but the combinations of the oxygen of the blood are not carried far enough to produce the final result of the normal oxidation in the healthy organism, and in addition to the formation of leucine and tyrosine, and of various nitrogenous substances, the composition of which is not perfectly known, *fat* results from these changes, which not only imparts an oily appearance to the blood, but also from the arrest of the oil globules in the capillaries infiltrates the textures of the liver, heart and spleen, and induces in these organs fatty degeneration.

CHAPTER IX.

BLACK VOMIT OF YELLOW FEVER.

The following observations on the black vomit of yellow fever, are the results of labors begun in 1856, and pursued, in various portions of the Southern States, up to the present time.

I have shown, by numerous careful post-mortem examinations, and by analysis of the blood, black vomit, and urine during life, in various stages of the disease, that:

During the active stages of yellow fever, profound changes take place in the organs and tissues, and especially in the kidneys, heart and liver; and oil and granular fibrinous or fibroid matters (altered albumin and fibrin), transude through the capillaries and fill up the cells and excretory ducts, and arrest or impair the functions of these organs.

The liver of yellow fever does not present the soft friable condition characteristic of true fatty degeneration. The jaundice resulting from the suppression of the excretory function of the liver, would appear to be due to the same causes which induced the suppression of urine, namely, to the deposit of oil and fibrinous matters in the excretory structures of the kidney and liver.

The heart, in yellow fever, appears to be as fully permeated with oil as the liver; in the latter organ, however, a large amount of the oil is inclosed within the liver cells.

Yellow fever is not only attended with what might be called "acute fatty degeneration," but also with such profound alterations of the fibroid elements of the blood as to permit the transudation of the altered fibrin and albumin, through the walls of the capillaries, into the excretory tubes.

Black Vomit.—This ejection of altered blood from the mucous membrane of the stomach, during the period of calm or depression, although not absolutely characteristic of yellow fever, as it may occur in other diseases, is still of so frequent occurrence in yellow fever as to demand the most careful consideration, both as to its nature and origin.

The character of the matters vomited during the progress of yellow fever varies in different stages of the disease, and with the character, relative mildness and severity, and the progress of the changes in the blood and organs.

While yellow fever is characterized, in common with several other diseased states, by an irritation of the gastric mucous membrane, the peculiar nature of the vomited matters does not rest entirely upon the congestion and irritation of the mucous membrane of the stomach, but is influenced to a greater or less extent by the changes of the blood, liver, kidneys and nervous system.

The vomiting in yellow fever may also be regarded, to a certain extent, as salutary, and as an effort for the elimination of certain excrementitious materials from the blood. In some cases, the first effects of the black vomit may seem to be salutary; the tongue improves in appearance, the febrile heat abates, and if it were not for other profound changes in the blood, liver and kidneys, lying back, as it were, of this almost universally fatal symptom, beneficial results of the most important character might flow from the relief afforded by the removal of a certain amount of excrementitious matter, as urea and ammonia from the blood.

The first ejections of the stomach of a yellow fever patient consist most generally of the ordinary secretions and contents of the stomach, then follow vomiting of a mucoid fluid, frequently at first tinged with bile, the reaction of which varies in different cases, being alkalin in some and acid in others.

After the first vomiting the stomach may remain tolerably quiet until the subsidence of the fever on the third or fourth day, when, without any premonitory symptom of nausea, the stomach, on any trifling provocation, may eject a quantity of clear, pale, almost limpid and slightly acid, opalescent fluid. At this period the disease may terminate, or make no farther progress, as if this elimination was similar to the perspiration of intermittent fever, the whole ailment vanishing at this stage. If the vomiting continues and passes on to black vomit, it becomes first streaked with dark flocculi of altered blood; the reaction, in many cases, changes from the acid to the alkalin, and careful chemic examinations have convinced me that this change in the reaction of the black vomit was due to the elimination by the gastric mucous membrane of urea, and its conversion into ammonia.

The acid reaction of the yellow fever vomit is due not to the presence of any peculiar acid, but to several, as the phosphoric acid, existing in the form of acid phosphates, acetic and hydrochloric acids. The degree of the acidity will also vary with the character of the fluids and solids taken into the stomach; thus, if much sugar be taken, the vomited matters will be much more strongly acid, and the presence of this substance will also determine, to a great extent, the presence and development of certain fungi, as the yeast plant.

The rapid generation of torulæ, as well as the effervescence of the black vomit, in certain cases, is referable chiefly to the presence of sugar in the aliment, and in the tea and coffee drank.

I have observed various forms of coniomycetous fungi in the black vomit of yellow fever, but I have by parallel experiments, and microscopic examinations of urine and various other organic liquids, failed to find any distinct species characteristic of yellow fever. If the black vomit be allowed to stand for days and weeks, and be carefully examined at intervals with the microscope, we see various forms of the coniomycetous fungi, as torula, sacchari or cerevisiæ, cryptococcus, and penicillium glaucum; and of the hyphomycetous fungi, as oidium lactis, and oidium albicans; and of the physomycetous fungi (mucor); and of the torulaci. Upon one occasion, during these investigations, I submitted one of these fungi from the black vomit of yellow fever to the observation of an excellent microscopist of this city, Mr. Kinttschmitt, and the following is his reply:

New Orleans, Nov. 10, 1873.

My Dear Sir:—I have subjected the filtrate from black vomit, which you recently placed at my disposal, to a very careful microscopic examination and comparison with other fungoid growths, of which I have several prepared slides in my possession.

The fungoid growth developing in the filtrate of black vomit has certain points in common with torula, oidium and mucor, and if torula be a submerged form of penicillium, also to the latter fungus; it also resembles the filamentous development of bacteria, the same as the fungoid growths developing in a solution of gamboge. The diagram which Virchow furnishes of the aspergillus fungus discovered by him in the human lung, has a strong likeness to the black vomit fungus.

There is a doubt, however, in my mind, that yellow fever is not produced by a specific fungus; but the fungus, if produced in the human body at all, is produced by the sickness itself; but it is most likely that the fungoid growth only makes its appearance after the vomit has been ejected from the stomach.

The slides on which the foregoing remarks are based, had been prepared on 2d inst. On examining them again yesterday, the fungus has in a measure disappeared, and granular masses seem to have taken its place. I remain, my dear sir, very respectfully,

P. Kinttschmitt.

The specific gravity of black vomit, as determined by weighing, with the specific gravity bottled, varies from near the standard of distilled water to near that of blood.

It is now fully admitted that black vomit is not entirely confined to yellow fever, and that it is chiefly the secretion of the mucous membrane of the stomach, and the matters introduced from without as food, mingled with the blood which oozes slowly into this viscus, from the mucous surfaces denuded of mucous epithelium.

But in most cases, black vomit is something more; it is to a certain extent an excrementitious product, containing urea and carbonate of ammonia.

The kidneys are more or less affected in every case of yellow fever; when they act continuously and freely, the blood is freed of bile and urea, and black vomit more rarely occurs than in those cases in which their functions are arrested, and the mucous membrane of the stomach assumes the excretory function.

Black vomit is due to several causes:

1. To the direct irritation and structural alteration of the gastric mucous membrane, by the poison of yellow fever. This poison is most probably received into the blood and acts in this manner, or through this medium upon the gastric mucous membrane, for we find cotemporaneous changes taking place in the heart, liver and kidneys; and these changes would most probably succeed the gastric irritation, if the poison was received in food or drink, primarily by the stomach.

2. To the structural alterations of the blood, and especially to the marked diminution of the fibrinous element, which appears to sink to a lower figure than in any other known diseased state. It is well known that the diminution of the fibrinous element, below a certain standard of health, so deranges the capillary circulation as to lead to congestion, alteration of nutrition and secretion, and passive hemorrhages. The hemorrhages and effusions of scurvy can only be referred, with reason, to the changes of the blood, and especially of the fibrin.

3. To suppression of the action of the kidneys, and retention in the blood of urea and other excrementitious products, and the elimination of urea, and carbonate of ammonia and ammonia, by the gastro-intestinal mucous membrane.

4. To the direct irritant action of the ammonia, and excrementitious materials eliminated vicariously,

upon the mucous membrane of the stomach and intestines.

Bernard and Frerichs long since explained, by experiments, what we observe in those cases of yellow fever attended with urinary suppression. Thus the former experimenter found that a dog, which had a fistulous opening in the stomach, passed daily with his urine about 93 grains of urea and uric acid, and yet during the succeeding twenty-four hours after the removal of the kidneys, the blood drawn from this animal exhibited only a mere trace of these constituents, but urea in abundance was detected in the gastric juice withdrawn through the fistula; and after remaining for some time in the stomach and intestine, the urea changed into ammoniacal salts, and the gastric juice was secreted continuously and not, as in the normal condition, only after a meal. Not only were the manner of formation and chemic constitution of the secretion of gastric juice altered by this vicarious excretion of the main constituent of the urine, but the mucous membrane was structurally altered, and became disqualified not only for the performance of this eliminative action, but also for the elaboration of its normal secretion.

In yellow fever, the suppression of the action of the kidneys follows immediately after, or may even begin in the midst of a devastating fever, attended with repeated alteration and chemic change of the elements of the blood, and the formation of large amounts of urea and other excrementitious matters, and the work suddenly thrown upon the already weakened and altered stomach is far greater than when the kidneys are amputated in a healthy dog, or when their sudden suppression is from the action of cold. Bidder and Schmidt have shown that the digestive powers of the gastric juice are weakened, if it be mixed with any considerable quantity of saliva, in consequence, as they suppose, of the neutralization of the free acid by the alkali of the saliva; and they also found that the addition of bile to the normal gastric juice entirely suspended its digestive property, although the mixture still exhibited an acid reaction. Bernard, Bidder and Schmidt found that gastric juice, secreted with urea, sooner or later not only became alkalin, but also lost its power of converting albuminous matters into assimilable forms; and Lehman also found that the digestive power of the gastric juice was much impaired by the addition of alkalin salts, or by saturating the fluid with peptones or other organic substances, either nitrogenous or non-nitrogenous.

In yellow fever, when there is an impairment or suppression of the function of the kidneys, we not only have a combination of these various causes, producing derangement of the gastric juice, but we also have the destruction of the fibrin of the blood,inducing passive hemorrhages from the congested and altered gastric mucous membrane, and at the same time such an elevation of temperature as is most favorable to the rapid decomposition of the contents of the enfeebled stomach and intestines.

5. To the irritant and nauseating effects of the bile retained in the blood. The bile retained in the blood, without doubt produces its characteristic effects upon the nerves supplying the stomach, producing nausea and vomiting.

6. To the degeneration of the excretory and secretory cells of the gastro-intestinal mucous membrane, attended with or characterized by the deposit of granular fibroid or albuminous matters and oil globules in the secreting cells, and in the walls of the smaller blood vessels or capillaries.

7. To the capillary congestion of the gastro-intestinal mucous membrane, similar in all respects to the intense capillary congestion which characterizes all the organs and tissues in this fever.

The chief causes of black vomit therefore are: the direct irritation of the gastric mucous membrane; intense capillary congestion, in consequence of the morbific action of the poison of yellow fever, and its products, upon the vasomotor system of nerves; suppression of the functions of the liver and kidneys, and the retention in the blood of bile and urea, and the elimination of urea from the gastro-mucous membrane, as such, and in the state of ammonia and carbonate of ammonia; and the direct irritant and solvent effects of ammonia and carbonate of ammonia, upon the gastro-intestinal mucous membrane and the effects of the urea, ammonia, and other constituents of the metamorphosis of the tissues upon the blood; the alterations of this fluid by the changes excited by the yellow fever poison, and the destruction and alteration of the fibrinous element.

When careful sections of the kidneys were made with Valentin's knife and examined under the microscope, the Malpighian corpuscles and tubuli uriniferi were filled with oil globules and granular fibroid matters, which appeared to be modifications of fibrin and albumin. The excretory cells of the kidneys also contained oil globules and granular matter.

The poison of yellow fever appears to act in an analogous manner to the agent producing smallpox, or the poison of certain reptiles, which alter the constitution of the blood, and lead to the formation of altered albuminous and fibrinous products from the blood; and which in the case of yellow fever transude into the hepatic ducts and urinary tubes, and thus cause suppression of the urinary and biliary secretions.

When in any case of yellow fever the function of the kidneys is arrested, a fatal result necessarily ensues, not only from the retention of the urinary constituents, but also from the retention of the bile.

As long as the kidneys perform their normal function, the retained bile will be continuously eliminated; but as soon as these organs cease to act, the bile, as well as the urinary excretion, is retained in the blood, and certain nervous disturbances are induced, as dullness of the intellect, uremic convulsions, and in some cases violent agitation of the muscles resembling tetanic spasms.

Black vomit, therefore, is an *effect* or *result* of preceding actions or changes, and is not a cause; it is an error therefore to search, either by chemic means or by the microscope for the *cause* of the disease in one of its *products*.

Black vomit, from its great amount, may be one of the causes of death; but as it is, in many cases at least, the result of an effort on the part of the living organism to eliminate certain materials from the blood, it may be to a certain extent salutary; but it is in all cases, only secondary to the preceding changes in the blood, heart, liver and kidneys.

In the preceding chapters we have given the practical results of the microscopic and chemic examinations of the black vomit in various stages of yellow fever; similar facts will also be presented in the succeeding chapter on the changes of the urine; the following cases are presented in this connection as direct demonstrations of the generalizations just recorded:

Case 1.—Yellow fever, jaundice, suppression of urine, black vomit, death. G. G., age 22, laborer; native of Canada. Entered Charity Hospital Oct. 18, 1873. Attacked with yellow fever October 14. Has been driving a street car, and has resided on Napoleon Avenue near the Mississippi River, in one of the so-called infected districts. Has resided in New Orleans four years; states that his attack began with "universal pain all over the body," which was greatest in the forehead and lumbar region. Fever began with chilly sensations. October 18, evening; has just entered the hospital. This is the fourth day of the disease. Pulse 100; respiration 30; temperature of axilla 105.5 F. Tongue very red at tip and edges, and coated with yellow fur in the center. Conjunctiva yellow and congested. Surface of the entire body yellow, with marked capillary congestion. Gums red. No appetite. Body emits a disagreeable odor. Patient restless and nervous, and although answering questions slowly appears to be in an unnatural state. Complains of pains in all parts of his body.

October 19, 8 A.M., tongue very red at tip and edges and furred in center. Gums very red. Conjunctiva of eyes congested and yellow; body deeply jaundiced, with great peripheral capillary congestion. Eyes have a wild restless expression. Stools light colored. Patient has passed no urine and his bladder is not distended. Restless, uneasy, sleepless with great mental hebetude and confusion. Pulse 90; respiration 26; temperature of axilla 103.10. 8 P.M., intellect confused and stupid; tongue red at tip and edges. Patient very restless, tries to get out of bed incessantly and says that he wishes to go home. Face expressive of sorrow and grief. No urine has been obtained during the night or day; complete suppression. Pulse 90; respiration 26; temperature of axilla 102. Black vomit runs from corners of the mouth and appears to regurgitate from the stomach without any effort on the part of the patient, who continued very restless during the night and died at 6:30 A.M., October 20. Black vomit continued to run out of his mouth up to the time of death.

Autopsy, at 9 A.M., October 20, three hours after death. Exterior of body still warm. The warmth was diffused over the entire body. Face and limbs full and round. Surface of skin greenish-yellow; dependent portions of neck, face, trunk and extremities mottled. Black vomit running in streams from both corners of the mouth. When the body was turned on its side in placing it on the table, a considerable amount of dark blood or black vomit poured out of the mouth. The ease which the black vomit poured out of the mouth indicated great relaxation of the muscular coats of the esophagus and stomach. Immediately after opening the thorax, I introduced the bulb of a delicate thermometer into the right ventricle of the heart, and found the temperature to be 100.5 F. It is not probable that any great degree of heat had been lost during the short period of time which had elapsed after death. Dependent portions of lungs congested, otherwise normal. Muscular structures of heart apparently greatly congested, giving the surface of the organ a deep color, as in malarial fever; but when the walls of the heart were cut through and the organ carefully washed under a stream of water the muscular structures presented the characteristic yellow color of yellow fever. Muscles of heart softened and readily crushed under the fingers. Under the microscope the muscular fibrillæ of the heart were found to contain numerous oil globules and much yellow granular matter; the oil and granular matter were, however, not so abundant as in cases in which death took place at a later stage. Cavities of heart distended with dark liquid blood; also a small golden-colored fibrous clot. Stomach distended with about one quart of dark, grumous, fluid black blood, or black vomit. Mucous membrane of stomach greatly congested and mottled, presenting deep purplish spots or ecchymoses, and from these portions the black vomit appears to have flowed from the congested and ruptured blood vessels. Small intestines contained blood.

The liver was congested, presenting a dark mottled appearance on the surface; many yellow lobuli, however, were readily discerned upon the surface, resembling in all respects the yellow color of the liver in yellow fever. The congestion of the capillaries of the liver was so great, however, that the organ presented more nearly the hue of some recent fatal cases of malarial fever. When thin sections of the liver were made, and the excess of blood gently pressed out and the slices washed under a stream of water, the color resembled in all respects that of yellow fever. Liver loaded with oil globules and yellow granular matter. Liver cells contained oil globules and were of a distinct yellow color.

When the great excess of blood with which the kidneys were congested was washed away, these organs presented the usual color of yellow fever. Sections with Valentin's knife, viewed under the microscope revealed the Malpighian corpuscles and tubuli uriniferi, filled with detached cells, oil globules and yellow granular matter. The urinary bladder contained about two fluid ounces of urine, which represented the amount excreted during the last thirty-six hours of life. Detected no coloring matter of bile in urine. Acid reaction. Slightly turbid. Light yellow color. Urine contained albumin, urinary cells from excretory tubes and from pelvis of kidney, and casts of tubuli uriniferi. Examination of black vomit from the stomach. Reaction acid; contained numerous colored blood corpuscles, masses of hematin, oil globules, granular matter, cells from mucous membrane, casts of gastric glands, and broken capillaries. Sp. gr. 1016.7. Upon careful analysis, the black vomit contained urea and ammonia, acetic and hydrochloric acid. No hydrocyanic acid was present. The hydrochloric acid loses to a certain extent, for it could be driven off by heating the black vomit gently in a beaker on the sand bath, and its fumes reddened litmus blue, and decomposed nitrate of silver. When thrown upon a filter, the filtrate presented a brownish-red yellow color. Sp. gr. of filtrate of black vomit 1013.2. When the filtrate of the black vomit was treated with nitric acid the albumin coagulated, but the coagulum re-dissolved in boiling. I was unable to detect the biliary coloring matter in the filtrate of the black vomit. Alcohol added to the filtrate coagulated the albumin. Sp. gr. of black vomit 1016.7. Sp. gr. of filtrate from black vomit after removal of blood corpuscles and organized bodies 1013.2.

Solid residue in 1000 parts of black vomit	56.15.
" " " " " " filtrate from black vomit.	46.60.
Hematin, blood corpuscles and organized elements in 1000 parts of black vomit	9.55.

When the filtered liquid from the black vomit was set aside, a delicate fungoid plant resembling the torula was developed. This delicate fungus had certain points in common with torula, oidium or mucor; and if torula be a form of penesedium, also to the latter fungus. It also resembles the filamentous development of bacteria. A similar fungus has been observed developing in gamboge.

Case 2.—Yellow fever, black vomit, death. John Sullivan entered Charity Hospital Oct. 8, 1873, about fourth day of disease, great congestion of capillaries of face, neck and extremities, jaundice, hiccough, nausea, pain in head especially in frontal region. Pulse 82, respiration 28, temperature of axilla 98.2. October 9, 8 A.M., pulse 72, respiration 28, temperature 97.1; black vomit. The liquid ejected in abundance is of low specific gravity and strong acid reaction and contains numerous dark specks and flakes resemblihg coffee grounds. Hands feel cold. Great congestion of peripheral capillaries. Jaundice; conjunctiva of eyes congested and yellow; tongue red at tip and edges. Gums very red with some oozing of blood. Patient says that he has pain everywhere. Urinary excretion suppressed. 8 P.M., pulse 80, respiration 30, temperature 99.2. Spits up black vomit all over the bed.

October 10, 8 A.M., pulseless, respiration 40; temperature 92.2. Suppression of urine. Patient restless and delirious. Spits up black vomit which resembles black liquid blood. Died at 10 A.M. Temperature of axilla at moment of death 99.2. There was no rise of temperature after death. Examination of black vomit; clear straw-colored liquid, holding in suspension dark coffee-ground-looking flocculi and flakes resembling coffee grounds, which settled to the bottom of the vessel, when the liquid was allowed to remain at rest. Reaction strongly acid. The reaction was found upon analysis to be due to acetic and hydrochloric acids. Under the microscope, I observed broken capillaries, cells of mucous membrane of stomach, blood corpuscles and angular masses of hematin and cells of the gastric mucous membrane stained with hematin. Numerous bacteria and rotating animalculæ, about one ten-thousandth of an inch in diameter rapidly appeared and increased in numbers. Specific gravity of black vomit 1004. When filtered, the colored blood corpuscles, masses of hematin, and mucous cells remained on the filter, and a clear, light, straw-colored liquid passed through. The filtered liquid gave a heavy precipitate with nitrate of silver, and a mere turbidness with the chlorid of barium. Nitric acid produced no precipitate in the filtered liquid. The graded solution of the nitrate of the per- (red) oxid of mercury used in the analysis of the urine after the method of Liebig produced a heavy white precipitate from the presence of urea. The usual process for urea in the blood and organic liquids revealed the presence of urea. This specimen of black vomit was collected October 9 at 10 A.M. Upon carefully tasting the black vomit I found it to be decidedly bitter like bile, although I was unable by chemic tests to detect either the coloring matter or the acids of bile. The black vomit became thicker toward the close of life, and more nearly resembled dark grumous blood. During the last thirty-six hours of life, this patient emitted a foul stench, and presented a horrible appearance, with the dark filthy stains of black vomit on the bed-clothes, and even the sides of the walls; streams of black vomit running from the corners of his mouth, and immense swarms of flies hovering over and around his bed.

Post-mortem examination: results similar to those recorded in many other cases of yellow fever already presented in detail.

Case 3.—Yellow fever, black vomit, death. Daniel McMonagale, age 18, native of Ohio, left his native State two years previous to the present attack; resided in Memphis and Vicksburg, and has been in New Orleans two weeks. Has had chills and fever at various times during the past two months. Entered Charity Hospital evening of Oct. 11, 1873. I saw this patient for the first time on the morning of October 12, 8 A.M. At this time, although not actually delirious, is unable to give a full and accurate account of his present attack. Surface of body covered with petechiæ. Gums red. Tongue very red at tip and edges. Body of tongue coated with light yellow fur. Tenderness on pressure of epigastrium. Conjunctiva of eyes injected. Center of tongue cracked, with dark blood oozing from the denuded surface. Conjunctiva of eyes and surface of body present a distinct yellow color. Nausea; pulse 110, respiration 22, temperature of axilla 104.6. 8 P.M., pulse 113, respiration 26, temperature of axilla 105.5. The pulse has increased two beats, the respiration four, and the temperature of the axilla, 0.9 F. Has ejected the matters contained in the stomach, with some flakes and streaks of dark blood. A small amount of urine was collected during the day which contained bile, albumin and urinary casts. The urinary secretion is scant and evidently diminished in amount. Injection of ice-cold water into the rectum was ordered; ice bag to epigastrium, and water charged with carbonic acid, iced milk and fragments of ice internally.

October 13, 8 A.M., pulse 110, respiration 29, temperature 104.5. Dull and stupid. Has thrown up black vomit in considerable quantity. The injection of the ice-cold water appeared to be beneficial and to increase the flow of urine. 8 P.M., pulse 112, respiration 34, temperature of axilla 104.7. Continue ice-cold injections to rectum and treatment. October 14, 8 A.M., pulse 116, respiration 35, temperature 103.3. Delirious; jaundice; great capillary congestion; urine scant. During the night the patient discharged black vomit over his bed and against the sides of the wall. At the present time, spits up black vomit against the wall, like tobacco juice. The black vomit appears to come up without any effort. The nurse found it almost impossible to collect the black vomit for microscopic and chemic analysis. No urine has been collected since the first day, although the nurse states that the patient has passed it in bed. The injection of the rectum with cold water appeared to produce beneficial effects upon the congested and embarrassed kidneys. 8 P.M., pulse 130, respiration 38, temperature of axilla 103.3. Great capillary congestion, conjunctiva and surface of a yellow mottled appearance. Petechiæ have continued upon the surface. Notwithstanding the increase in the rapidity of the pulse and respiration, the temperature has fallen 2.2.

F. since October 12. Died October 15 at 6 A M. This patient was delirious for thirty-six hours before death.

Autopsy three hours after death: exterior of body of a golden jaundiced color; lower portions of a deep purplish mottled appearance. Black vomit issuing from corners of mouth and streaming down the neck. Limbs, trunk and face full and plump. Blood issuing from both ears. Thorax: lower portions of lungs congested, otherwise normal. Heart: muscular fibers presented a yellow appearance and were much softer than in healthy hearts, and in the heart of malarial fever. Under a magnifying power of 450 diameters the transverse striæ of the muscular fibers were found to be indistinct, and to be filled with granular matter and small oil globules. The granular matter and small oil globules appeared to be deposited around as well as within the muscular fibrillæ. The blood from the cavities of the heart was carefully collected; it was still warm, and fluid. Upon standing a few minutes in the glass vessel it coagulated. Under the microscope the vast majority of the colored blood corpuscles presented a normal appearance; a few presented a crenated stellate appearance. The blood corpuscles ran together, forming rouleaux as in the blood of inflammation. The fibrin was diminished in amount—the clot was voluminous and soft, but the fibrin was in larger amount, and the clot was more firm than in those cases in which there had been complete suppression of the urine. Upon careful examination, with a high power, 1-18 of inch, I discovered no bacteria, animalculæ nor fungi in the blood. The blood, however, contained numerous oil globules. Reaction of blood neutral or very slightly alkalin. Upon standing for a few hours, golden-colored serum was pressed out of the clot, which also contained colored corpuscles. At the end of three days the clot still remained undissolved. The serum was less deeply colored than in those cases in which there was complete suppression of urine. The liver upon the exterior presented a yellowish mottled appearance. This appearance was also observed when sections of the organ were made, and appeared to be due to the congestion of the hepatic capillaries within each lobule. The periphery of each lobule, including the portal system of capillaries, was of the usual yellow color, and the oil appeared to be accumulated in largest amount in this portion of the lobuli. Some portions of the liver were without any marked capillary congestion of the hepatic venous capillaries, and presented a uniform yellow appearance. This liver in a general view, presented more nearly the Spanish brown color of the liver of health than in many cases of yellow fever; and this depth of color was due to two causes: 1, to the great congestion of the central capillary (hepatic capillaries) of each lobulus; and 2, to the presence of dark granular masses of hematin, scattered chiefly through the portal system of capillaries, and which had evidently been deposited during the preceding attack of malarial fever. The blood was much brighter in color in the capillaries of the liver than in malarial fever. The blood from the cavities of the heart and from the blood vessels of the liver also changed to a bright arterial hue. The voluntary muscles, as those of the thorax, presented a brilliant hue when exposed to the atmosphere. The muscular fibers of the heart on the contrary presented a distinct yellow color like that of the liver. The gall bladder was entirely empty, kidneys of a yellowish color and congested. Sections of the kidneys with Valentin's knife were carefully examined with magnifying powers ranging from 1-2 to 1-18 inch objective. The Malpighian corpuscles and tubuli uriniferi contained detached cells, oil globules and yellow granular matter. These matters have accumulated to a much less extent, however, than in the kidneys of those cases of yellow fever in which there had been complete suppression of urine. Urinary bladder distended with golden orange-colored urine. Specific gravity 1012; reaction acid; urine contained albumin in considerable amount with detached cells and casts; no grape sugar. Alimentary canal, mucous membrane of stomach congested, with denuded patches, in which the ends of broken vessels filled with dark liquid blood were plainly seen, especially when low magnifying powers were employed. The stomach contained eight fluid ounces of thick, grumous, almost black liquid blood or black vomit. Under the microscope, the contents of the stomach contained numerous blood corpuscles variously altered in their forms, masses of hematin, mucus corpuscles, broken capillaries, detached epithelium from mucous membrane and granular casts of the glands of the mucous membrane of the stomach. No animalculæ or vegetable organisms were observed in the black vomit. Some oil globules were observed, which were referred to the milk which the patient had taken before death. The black vomit emitted a foul putrid odor as of decomposed blood. Reaction of black vomit slightly acid. Spleen, enlarged, softened and exhibited the marks of preceding malarial fever.

The foregoing investigations occupied my time fully for twelve hours without intermission. I determined to institute a series of experiments with black vomit upon living animals, and at 6 P.M., Oct. 15, 1873, I instituted the following experiments:

EXPERIMENTS UPON LIVING ANIMALS WITH THE BLOOD FROM THE HEART, AND THE BLACK VOMIT FROM THE STOMACH, OF YELLOW FEVER CASES.

Experiment 305.—With a small "subcutaneous" syringe, I injected beneath the skin of a healthy puppy, about thirty drops of blood taken from the heart of a yellow fever patient three hours after death. No ill effects were observed.

Experiment 306.—In like manner I injected beneath the skin of an active guinea pig about the same quantity of blood from the yellow fever heart. The next day, Oct. 16, 1873, the guinea pig appeared lively and ate its food. October 17, the animal appears sluggish and refuses food. October 18 animal feeble;

moves with difficulty, and is evidently ill from the effects of the injection. October 19, 5 A.M., I found the guinea pig dead, cold and stiff.

Post-mortem examination.—Body emits a disagreeable odor; cellular tissue of skin and surface discolored and greatly congested around the point of the injection of the yellow fever blood. Cellular tissue and skin softened in those portions around the area of injection. Under the microscope the fluid from these portions of the cellular tissue was found to contain bacteria and revolving animalculæ. Cavities of the heart distended with dark, almost black, loosely-coagulated blood. Blood changed rapidly to the arterial hue upon exposure to the atmosphere. Under the microscope the blood corpuscles presented no peculiar alteration. Liver congested. The microscope revealed no accumulation of oil in the textures of the liver. Liver cells distinct but pale. No bacteria, or animal or vegetable organisms were observed in the blood of the heart, or in the blood and structures of the liver.

Experiment 307.—Into the subcutaneous tissue of a large, healthy and active male guinea pig, I injected about thirty drops of black vomit, taken from the stomach of the yellow fever patient three hours after death. The black vomit thus carefully injected into the cellular tissue caused the death of this animal in six hours.

Post-mortem examination.—Diffused redness, and great capillary congestion of cellular tissue beneath the skin. Congestion greatest in the immediate vicinity of the injection. Body emits a foul putrid odor. Immediately around the point where the black vomit had been injected dark blood had been effused, and the textures presented precisely the appearance of those wounded and poisoned by the fangs of the rattlesnake or copperhead. The cavities of the heart were distended with dark loosely coagulated blood. No animalculæ, bacteria or fungi, or algæ discovered in the blood. Decomposition rapid; and although the animal was examined almost immediately after after death, the odor was disagreeable and resembled that of the black vomit.

Experiment 308.—I injected into the subcutaneous tissue of a large healthy guinea pig, about thirty drops of black vomit which I had preserved from a case of yellow fever, ejected shortly before death on Oct. 9, 1873, six days before the present experiment. Death caused by the black vomit in six hours.

Post-mortem examination.—Results similar in all respects to those recorded in the preceding experiment; intense congestion of cellular tissue around locality of injection of black vomit; no animalculæ or vegetable organisms in blood; putrefaction rapid and marked by foul odor.

Experiment 309.—I injected into the subcutaneous tissue of a guinea pig about thirty drops of putrid blood, which I had extracted from the cavities of the heart of a subject who had died six days before, on October 9, in a "*congestive*" malarial chill. Death caused by the putrid malarial blood in eight hours.

Post-mortem changes similar to those recorded in experiments third and fourth.

The preceding experiments are of importance in establishing the fact that black vomit, taken from the stomach immediately after death, or ejected during life in yellow fever, will, when injected into the subcutaneous tissue, produce as deadly and as rapidly fatal results as putrid blood.

We deduce the following practical conclusions:

1. Black vomit, when absorbed into the circulatory system, may act as a deadly septic poison.

2. As the capillaries of the stomach in yellow fever are often ruptured, and the epithelium of the mucous membrane denuded, it is possible that in some cases the absorption of black vomit, especially after it has undergone putrefactive changes in the stomach, may be an important cause of the fatal issue.

3. If remedies could be used which would prevent putrefactive changes in the black vomit and render it

comparatively inert, a certain proportion of cases might be rescued, after the appearance of black vomit.

The preceding experiments and reasoning led me to employ the sulpho-carbolate of sodium, in doses of 20 grains, every four or six hours in the treatment of yellow fever. I have used the sulpho-carbolate of sodium in about forty cases of yellow fever, with satisfactory results. I find that it is readily borne by the stomach at all stages of the disease; that so far from exciting nausea or vomiting, it often modifies and arrests these distressing symptoms. It appeared in some cases to arrest the decomposition of the black vomit. One case accompanied by a temperature of 107.1 degrees, and attended with black vomit recovered under its use, combined with ice water injections into the rectum. At some future time we hope to present statements of these cases.

Davaine claims to have made the following discoveries: after injecting beneath the skin of an animal's neck a single drop of putrid blood, the surrounding tissue became extensively infiltrated, and death soon followed with symptoms of septicemia. The blood of this animal was then employed to inject other animals in a similar way, and was found to be more poisonous than the original putrid blood. A third and fourth animal was then injected, each with the blood of the preceding one, and in this way twenty-four in all were experimented on. The results seemed to show that the toxic power of the septic material increases by dilution.

Stricker, in a series of experiments undertaken to test these statements, inoculated 25 animals with healthy, 23 with putrid, and 73 with blood diluted by transmission. Of the first series, 4 died. Of the second series 11 died. In experiments with diluted blood, when it had passed through 13 different animals, 53 of the 73 died. From this it appears that even the very small amount of 1-1250 of a cubic centimeter of poisonous material could cause death when injected hypodermically. Davaine's statement that organisms form in the blood and increase by every transmission, was not sustained by investigation. Stricker found numerous colorless bodies in the blood of these animals, but he did not regard them as organisms, but rather as protoplasmic bodies. The following facts he regards as ascertained:

1. That transmission greatly increases the injurious and fatal action of the putrid matter.
2. That the original disease was infectious, but that through inoculation it became contagious.
3. That though it be not proved yet it is probable that the special poison is a living contagion (*contagium animatum*) for such rapid proliferation is only possible in organized material.
4. That the poison is diffusible and is not destroyed by boiling (*Allg. Wein Med, Zietung*, 20, 1873; *New York Medical Record*, July 1, 1873, p. 311).

If it be true that the poison of yellow fever may be generated in human beings under certain conditions of the constitution, and more especially of the chemical and physical constitution of the blood and of the nervous and muscular structures, when subjected to the combined influences of heat and crowding in an impure atmosphere; if it be still further true that decomposition, both before and after death, is more rapid than in any other form of disease, and if this decomposition forms the most favorable condition for the rapid multiplication of bacteria and fungi, and other simple organisms; it is reasonable, in the light of the preceding experiments of Davaine and Stricker, to suppose that these organisms may become *carriers* of the poison, and may constitute an important medium of its dissemination from the original foci.

Magendie has shown by actual experiments on living animals that an excess of alkalinity in the blood, interferes not only with the freedom of the passage of this liquid through the capillary vessels and diminishes the property of coagulation, but also, when thus modified, the blood tends to penetrate the walls of the capillaries by imbibition, and to produce those disorders in the mucous membrane, which have long been known under the name of inflammation.

Magendie by injecting carbonate of soda into the veins of animals, produced edema of the lungs, with its pathognomonic signs at the outset, and cadaveric lesions when it causes death. Thus proving that when the blood becomes surcharged with an alkalin principle its serous matter increases in quantity, escapes from its vessels among the lobular ramifications of the lungs, distends and bursts them, and carrying with it the coagulable part, in a semi-liquid state, collects in irregular masses, which appear to be formed in large measure of fibrinate of soda and potassa.

On comparing the disorders caused by spontaneous excess of alkalinity in the blood, with those produced by injecting carbonate of soda into the veins, or by a frequent repetition of blood-letting, Magendie found the same symptoms and similar results.

If any substance possessing the property of combining chemically and of forming salts with the fibrin, such as fibrinate of soda, potassa, or ammonia, be injected into the veins of a living animal, the fibrin will lose its coagulability, and this change affects the blood generally, and although it may contain the usual proportion of fibrin, it becomes unfit for circulation, and stagnates in the capillaries and especially in the pulmonary vessels, and local lesions, as apoplexy, hemorrhage, or hepatization follow.

Magendie has in like manner, tested the effects of defibrinization upon the process of inflammation during the reparation of wounds. He selected a dog from whom he had successively removed several portions of fibrin. A longitudinal incision was made through the skin, and some depth of muscle in the anterior and middle part of the neck. The blood that escaped from the divided vessels appeared more liquid than usual; it did not coagulate on the blade of the bistoury. The lips of the wound were united by the twisted suture, and the animal left to himself. The animal survived the operation a few days only. An examination of the wound after death revealed the absence of any real adhesion by coagulable lymph, and the divided tissues were discolored, dry and hardly at all swollen.

The older writers were acquainted with the fact that alkalin salts prevented the coagulation of the blood, and John Huxham in his "Essay of Fevers," uses this fact to explain the changes of the blood in scurvy and putrid petechial fevers:

> "It is well known that volatile alkali salts mixed with the blood, when just drawn, or rather after it runs from the vein, keep it from coagulating and hinder it from separating into crasamentum and serum, as usual. The experiment is easy, and every one will find it true on trial. This very adequately resembles the blood drawn from the bleeding scorbutics, and also from most persons that labor under putrid petechial fevers, when the blood is drawn very early in the disease.
>
> "All humors of the body, actually putrefied, become a strong alkali, and putrid blood loses its consistency, and soon after its color, running into a yellowish dark-colored sanies. The blood drawn in some greatly putrid petechial fevers, hath had this appearance, and been observed actually to stink as soon as drawn, as well as the urine, as soon as made; so far was the putrefaction advanced, while even life was still subsiding. The surprisingly great and speedy corruption of bodies dying of pestilential fevers with spots, shows this likewise; I have known such a corpse air as much, as they call it, in seven or eight hours, as dead bodies commonly do in seven or eight days, and to leak out a most putrid sanies, from all the outlets of the body; which by-the-by, is a reason why persons dying of such fevers should be buried very soon."—"An Essay on Fevers," etc., by John Huxham, M.D., 1757, pp. 50, 51.

Andral has shown that in the diminution of the fibrin, relatively to the globules, we must recognize the grand condition of the blood favorable to the production of hemorrhages; and in his investigations he found the relations of these two factors so constant that he found it impossible not to regard the one as the cause of the other. In scurvy the proportion of globules is natural, or varies within narrow limits, while the quantity of the fibrin is diminished, and its coagulative quality is at the same time altered; and this condition is most favorable to the production of repeated and profuse hemorrhages.

The tendency of the formation of petechial and livid spots, during the progress of malignant fevers, in the Middle Ages, is referable chiefly to the scorbutic condition of most patients, at those times, when salt meat constituted the chief nutriment during a large portion of the year, and fresh vegetables were almost unknown. In this connection it should be remembered, that authors have declared that they have found an excess of alkalin matters in the imperfectly coagulated blood of persons who have died of low fevers or scurvy, attended with livid petechiæ and spots. An analogous result was announced by M. Frémy and Andral.

Without doubt the supposition is correct that the peculiar hygienic condition of the people of Europe, before the eighteenth century, caused them to be frequently attacked with diseases, one of whose principal elements, if not their starting point, was a state of dissolution of the blood. The dissolved and incoagulable blood, petechial and passive hemorrhages, so constantly described by authors, in their histories of epidemic fevers, in the Middle Ages, are most philosophically referred in a measure at least, to the scorbutic state of the blood of the patients, and to the bad ventilation and filthy habits of the times. In the epidemics which prevailed in Europe during the Middle Ages, it was common to observe gangrene, hemorrhage from various parts, extensive ecchymosis, or thousands of petechiæ covering the skin, while the symptoms of typhoid fever, and of the various contagious eruptive fevers, developed themselves with a high degree of intensity, and the greatest rapidity. These affections were the external manifestion of an internal condition of the blood.

Erasmus, the philosopher, who flourished toward the close of the sixteenth century, wrote that in his day, the inhabitants of London, were every year, from spring to harvest, attacked by a malignant fever. which committed the greatest ravages in that city, and especially among the poorer classes. "The supply of water," Erasmus says; "fails the inhabitants; they have to seek it at a great distance from the city; the river water is carried on their backs, and is so dear that the poor can not procure enough of it to wash themselves, and keep their houses clean. These houses are of wood, and very cold in winter, which makes it necessary to fill the rooms with straw. But as this can not be often renewed, it becomes spoiled and very injurious."

Grant who practiced medicine, and wrote toward the close of the last century, suggested the inquiry, whether the peculiar hygienic condition of the people of Europe before the eighteenth century, must not have caused them to be frequently attacked with diseases, one of whose principal elements, if not their starting point, was a state of dissolution of the blood. It is certainly remarkable that the observers of preceding ages, constantly speak of dissolved and incoagulable blood in their histories of epidemics.

Huxham in his "Essay on Fevers," has given a large number of observations, illustrating the great tendency to petechiæ and passive hemorrhages in the fevers of those times.

Andral in treating of the changes of the blood in scurvy, records the disappearance of this disease, and of the great febrile epidemics of the Middle Ages, and says:

> "We must therefore admit, that in consequence of the change in the nature of the influences which necessarily act upon men, the blood which receives before the solids the impression of the greater part of these influences, must present changes in its constitution proportioned to those undergone by the agents which operate upon it. It would appear then, that there must have been a time, when a very peculiar constitution of the blood engendered maladies, which in certain respects, may have differed from those now observed, and may not have required the same treatment.

And thus it is that at different periods of the existence of our race, and the diversity of influences to which it may be subjected, diseases of very different types may arise, and undergo changes in their essential nature, which are revealed to us by the specific character of their symptoms."

The changes of the blood in certain cases of yellow fever, may have been the *result* and not the *cause* of the aberrated nervous actions. If this be true, the petechiæ and spots, observed in a certain proportion of cases, should not be regarded as true cutaneous affections characteristic of the exanthemata.

It has been said that extreme agitation of the nervous system may deprive the blood of its power to coagulate. According to several observers, a similar effect may be produced by a strong moral emotion, a concussion of the brain, the destruction of a certain portion of the spinal marrow or a violent blow upon the pit of the stomach, affecting the nervous plexus of that region. If facts of this class were appropriately verified, they would doubtless be of the highest importance; for they would show that the nervous system exerts a powerful influence over the constitution of the blood, and that consequently a lesion of innervation may deteriorate the blood, just as an alteration of the blood may modify the nervous action. Professor Dupuy announced that by dividing the pneumogastric nerves in horses, the blood of these animals lost its property of coagulating; Dr. Mayer, on the other hand, having tied the pneumogastric nerves in living animals, found that uniformly the blood had coagulated throughout the entire pulmonary circulation. These facts, as well as those relating to the effects of lightning, excessive heat and excessive exercise in warm weather, require additional proof. It it be true that the changes of the blood may be in accordance with the portion of the nervous system affected, as in section of the pneumogastric and sympathetic, it may be inferred that the changes of the blood in yellow fever may vary within certain limits with the portion of the nervous system chiefly involved.

EXPERIMENTS ON THE EFFECTS OF PUTRID ANIMAL MATTERS.

One of the most remarkable phenomenon presented by animals into whose veins Magendie more than thirty years ago, injected putrid substances was what is termed inflammation of the intestines; that is to say, exhalation of a matter having the color of washings of flesh.

Various observers have pointed out the effects of certain animal substances in raising the temperature when injected into the circulation. Billroth and Hufschmidt, found that in all the cases in which putrid solutions or recent pus were injected into the subcutaneous tissue or into the blood, there was a rise of temperature in the rectum, which was considerable even within two hours after the injection, and reached its maximum in from two to twenty-eight hours; that the minimum exceeded the normal temperature by 1.6 C. (2.88 F.,) and the maximum, 2.2 C., (3.96 F.,) and that if the injection was only done once, a rapid defervescence generally set in, shortly after the acme had been reached; while on the other hand, after repeated injections death constantly occurred, generally with very high temperatures.

C. Weber has determined by similar experiments, the heat producing and inflammatory effects of pus, of fluids from inflamed tissues, and of pyemic and septicemic blood, and even of the blood of an animal merely suffering from simple inflammatory fever, when injected subcutaneously or into serous cavities.

Frese by numerous experiments, showed that the blood of animals suffering from any kind of fever, induced a rise of temperature, when introduced into the circulation of a healthy animal of the same species.

CHAPTER X.

EFFECTS OF PUTRID MATTER ON THE BLOOD.

M. Gaspard appears to have been one of the first experimenters with putrid matters. On June 19, 1809, he injected into the jugular vein of a small bitch, half an ounce of fetid liquid, arising from the simultaneous putrefaction of beef meat and dog's blood. On the instant the animal made many movements of deglutition, and very soon afterward experienced dyspnea, *malaise* and depression. She lay on her side refusing all food, and soon voided first her excrements, then her urine. In an hour's time, prostration of strength, gelatinous and bloody alvine discharges often repeated, dysentery, redness of conjunctiva; afterward, chest painful, belly hard and painful when touched; gradual extinction of strength; bilious, gelatinous and bloody vomitings. Death three hours after injection. On opening the body, yet warm, the lungs were engorged with blood and of a violet or blackish hue, with many ecchymosed or petechial spots, which likewise existed in the left ventricle of the heart, in the spleen, mesenteric glands, gall bladder, and even in the subcutaneous cellular tissue. The peritoneum contained some spoonfuls of a reddish serosity, but the mucous membrane of the alimentary canal was most affected. That of the stomach was slightly inflamed; that of the intestines, above all—the color livid, with black points, and covered with a gelatinous and bloody substance, resembling the lees of wine or the washings of flesh. In addition, this inflammation was accompanied with a slight thickening of the tissues, and possessed a hemorrhagic or scorbutic appearance.

On July 14, 1821, Gaspard injected into the right jugular of a large dog, two ounces and a half of a fetid liquid that had arisen from the fermentation for two days of cabbage leaves, at a temperature of 20 degrees R. It was thick, not at all acid and was mixed with an equal quantity of water. While injecting the animal often swallowed, and before long began vomiting, which was frequently repeated, and soon fell into a state of depression. Some hours afterward, great general uneasiness, pain in chest on pressure, respiration embarrassed, difficult and plaintive, appearance of pleuropneumonia, then vomiting anew and great depression all day. At the expiration of nine hours he had in the night a very fetid liquid stool, black as soot, analogous to the evacuations in melæna, and formed of a little excrement and mucus, with a great deal of apparently putrid blood. Some time afterward the dog had another stool, but it was merely muco-sanguineous. On July 15 (second day) depression more considerable, adynamia, recumbent on the side, vascillating walk, pulse small and febrile, ardent and seeming inextinguishable thirst, urine natural and abundant, respirations few and weak. During the day the pulsations of the heart would return at intervals with an extraordinary strength and noise, resembling what occurs in the highest degree of aneurysm, combined with hypertrophy of the organ. July 16, less depression, cessation of the disordered action of the heart, but still ardent thirst, refusal of food; fever and sometimes vomiting of drink. July 17, same condition. July 18, symptoms aggravated, extreme debility, walk tottering, excessive thirst, eyes red, inflamed and blear, nostrils swollen and filled with mucus obstructing the passage of air, mucous membrane of the mouth violet red and phlogosed. At mid-day, liquid stool of a whitish-gray color, mixed with grumous blood, of a purulent character and odor. Death during the night at the termination of the fifth day of the experiment.

Before opening the body, the skin, subcutaneous cellular tissue and muscles presented the same appearance as after death from asphyxia from want of air, and did not appear exempt from inflammation. Conjunctiva, pituitary and buccal membranes red or violet, and covered with a thick abundant mucus, lungs of a gluey feel, slightly phlogosed in some spots, but crepitant enough. The left ventricle of the heart presented many brown spots or sort of ecchymoses, penetrating even into the tissues; it was, besides, of the color of the lees of wine which contrasted singularly with the natural color which the right ventricle preserved.

The right ventricle was in part filled up with a hard albumino-fibrous concretion, with a yellowish-white hue like fat, very homogeneous, undistinguishable from the molecules of the injected liquid, weighing two and one-half grams, almost entirely free, and only adhering to the ventricle by a small attachment. This concretion with ramifications of the same color and consistency, extended into the pulmonary artery, and into the superior vena cava, and also into the azygos, axillary, and even to the right jugular. Probably it had been the cause of the violent pulsation of the heart. The esophagus and stomach were apparently healthy, but the mucous membrane of the intestines, and particularly of the duodenum, rectum, and a small portion of the small intestines was of a violet-red, colored chiefly in longitudinal wrinkles and irregular patches, which gave a parti-color to the outside of the intestines before they were cut open. Otherwise this inflammation was without thickening of the tissues, without ulcerations and much resembled ecchymoses or hemorrhage. In the duodenum were observed many kinds of open blisters, whence a large quantity of sanious blood was made to flow by pressure on the neighboring mesenteric vein. The internal membrane of the rectum was still more affected, and its mucous glands were very swollen and distinct. The intestine contained puriform matters, resembling those of the last evacuation. The other intestines contained mucus matter of a whitish-gray color, and very thick. The mesenteric glands appeared as if penetrated by blood and altogether inflamed. The gall-bladder, stained exteriorly by brown and violet spots, was filled with a black thick bile, as ropy as melted glue.

Gaspard also performed other experiments with putrid liquids, with similar results; and he demonstrated for the first time, that putrid liquids injected into the cavity of the pleura, etc., produced the same effect as injections into the veins (*Journal de Physiologie*, tome 2).

M. Magendie, fully appreciating the important facts that since medicine has existed, the pernicious influences of ponds, marshes, neglected harbors, and in general, all places, in which animal or vegetable matters were undergoing putrefaction, are recognized; and that men and frequently animals, inhabiting the neighborhood of these infected foci, were subject to serious maladies, which authors have designated under different names, such as the plague, intermittent fever, malignant fever, dysentery, cholera morbus, typhus, yellow fever, etc.; repeated with the greatest care the experiments of M. Gaspard, with an eye to their application to medicine, and the elucidation by the experimental method of the manner in which putrid animal or vegetable matters act upon the healthy individual. Magendie affirmed that the results of M. Gaspard were perfectly exact; and in addition he observed that different kinds of flesh have not the same activity in their putrefaction. The muscles of herbivorous mammalia appear less active than those of the carnivora. Putrefied oyster water did not cause very violent effects; but the deleterious matter *par excellence*, is putrid fish water; some drops of this water injected into the veins producing in less than an hour, symptoms which have the greatest analogy with those of typhus and yellow fever. Death usually ensued in twenty-four hours, and upon opening the body all the traces of a chemic alteration of the blood were discovered. The blood remained for the most part fluid; it had transuded through the walls of the vessels into the different tissues—particularly was it found to have traversed the intestinal mucous membrane. Blood as well as mucus accumulated in the stomach and intestines, where it presented all the intermediate hues between bright red and deep black.

Such experiments seemed to sustain the view that the healthy state of the blood in which its tendency to coagulation is very strong, prevents the transudation of the liquid through the walls of the smaller vessels. Magendie established that the same putrid matter, so deleterious when injected into the veins, has no bad effect when introduced, even in a strong dose, into the stomach or large intestines of animals; and that when putrid liquids were filtered, the animal which received the filtered liquid into its veins, experienced much less intense and much more prolonged pain than that which received the unfiltered liquid. An equal quantity of the same putrid water injected into the veins, or introduced with suitable precautions, into the divisions of the bronchia, do not produce the same effects. Injections into the lungs produce less serious consequences than injections into the veins. Magendie also subjected animals to the effluvia of putrefying vegetable and animal substances; and he thus sums up the results of these and the preceding experiments:

"From the preceding facts, it is seen that putrefied liquids when they are injected into the veins cause death, or effects which have the greatest analogy with those of yellow fever and typhus; that the prolonged respiration of putrid miasms produces death also, but in a period much longer and with symptoms which differ from the diseases I have just named. What can be the cause of such difference of the mode of action in the same substances? Why this diversity in their deleterious properties? It would be so much the more important to be able to answer these questions, as therein lies the whole difficulty, relative to the epidemic diseases which have recently occupied the public mind."

Among the conjectures which may be offered, there is one which merits particular attention. We may presume that different atmospheric conditions, and particularly temperature and moisture, ought to have a great influence upon the mode of action of putrid miasms. (*Journal de Physiologie*, tome 3; "Speculations on the Cause of Yellow Fever," by John Harrison, M.D., Professor of Physiology and Pathology in the Medical College of Louisiana, New Orleans; *Medical and Surgical Journal*, March, 1847, vol. III, No. V, pp. 568–573.) The results obtained by MM. Leuret and Hament (*Journal des Progrès des Sciences Médicales*, 1827, vol. V, pp. 1, 181), were similar to those of Gaspard and Magendie.

The more recent experiments of Dr. Andreas Högzes, of the University of Pesth, performed during the epidemic of cholera in that city in 1873, are of interest in connection with the present discussion of the effects of putrid and morbid matters on the blood.

Dr. Högzes endeavored to solve the following questions:

1. Are fresh choleraic discharges capable of exerting a deleterious influence on the organism, and in what form?
2. Does gastric and intestinal catarrh, artificially produced, increase the liability to be affected by choleraic discharges?
3. Is the air capable of carrying with it particles or choleraic evacuations, which act injuriously on the organism; and if this be the case, is there a difference between non-disinfected and disinfected discharges, simple diarrheal dejections and putrid fluids?
4. Are choleraic evacuations, when freed from elementary organisms, capable of affecting animals?
5. What parts of disinfected and of non-disinfected choleraic discharges, does a current of air carry with it? What is the further fate of these elementary

forms, if they fall on a soil favorable to their development, or on one that is neutral? In what way do they modify the action of the medium?

In his experiments, Dr. Högzes used choleraic evacuations that had been discharged an hour, or an hour and a half. The animals operated on were dogs and rabbits.

Six dogs of middle size were the subjects of experiments bearing on the first and second questions. In three, catarrh of the stomach and intestines was induced by the administration of the sulphate of copper and the subcutaneous injection of a few doses of croton oil; the other three were allowed to remain healthy. As soon as the vomiting and diarrhea had ceased in the first three dogs, Dr. Högzes administered to each of two dogs (one healthy and one diseased), fresh urine, intestinal evacuations, and vomited matters, from choleraic patients. Both dogs had intestinal catarrh (frequent vomiting and diarrhea). The dog with the sound intestinal canal was well on the third or fourth day; while one of those in which gastro-intestinal catarrh had been induced, died on the second day, having had constant vomiting and fluid evacuations; and the other two were not convalescent until the fifth or sixth day, and remained weak some time afterward.

To obtain an answer to the third question, Dr. Högzes kept rabbits exposed for some time to currents of air charged, 1, with non-disinfected; 2, with disinfected choleraic evacuations; 3, with ordinary diarrheal stools; 4, with the fluid of putrid meat. The oxygen necessary for respiration was supplied in abundance. The rabbit in each case was confined in a glass bell jar, placed in a well ventilated apartment; the bottom of this jar was airtight, and the air, charged with the evacuations or with the putrid fluid, passed in by one aperture at the top, and escaped by another. The air having been first made to pass through the fluid of which the effect was to be tested, was forced into the jar by a Bunsen's water apparatus, at the rate of 15 liters per minute. To prevent the air from escaping in its foul state through the ventilating tube, it was made to pass through cotton wool and sulphuric acid, and in this way it was possible, without danger of injuring the health of the operator to continue the experiment for any time.

Two rabbits in one of which bronchial catarrh had been induced by the inhalation of ammonia, were subjected for twenty-four hours to a stream of air charged with non-disinfected choleraic evacuation. While under the bell the animals had slight catarrh, did not eat and were depressed. In the third twenty-four hours, violent diarrhea set in, and both animals soon became cold and collapsed; the one in which bronchial catarrh had been induced, died first, and the other five hours afterward—both presenting the same symptoms. A small rabbit exposed for twenty-four hours in the same apparatus to air charged with choleraic discharges disinfected by carbolic acid, remained healthy. Another animal of similar size was kept without injury for twenty-four hours in a current of air saturated with ordinary diarrheal evacuations. A large rabbit, exposed for twenty-four hours to a stream of air, saturated with the fluid of meat that had been decomposing during five weeks, at first lay stupefied at the bottom of the apparatus, but immediately afterward rose up, and escaped without injury.

The experiments bearing on the fourth question consisted in the injection into the jugular veins of dogs and guinea pigs, in one case of choleraic discharges containing microscopic organisms, and in the other cases of the same discharges after filtration. With regard to the last question, Dr. Högzes conducted through fresh non-disinfected choleraic discharges a slow stream of previously purified air. The elementary forms brought over with the air current were received into two media—one neutral, distilled water; and one suitable for their development, Cohn's fluid. The fluids were drawn off by drops from the vessels, and examined with the microscope. Both distilled water and Cohn's fluid have no action when injected into the vessels; and the question was, whether their action would be changed by a certain amount of saturation. In a short time, a large number of elementary forms (chiefly bacteria), with which the cholera excreta were saturated, were carried over into the fluid, and in twelve hours produced a milky cloudiness in Cohn's solution. At the end of the second twenty-four hours the surface of Cohn's fluid was covered with a bluish-green shiny layer of cryptogamic matter, 2 centimeters thick, and the fluid itself assumed a peculiar smell, reminding one of decomposing fruit. The distilled water which remained quite clear, although charged with organisms, and the Cohn's fluid were injected into the veins of dogs and rabbits. The result was, that both the distilled water and Cohn's fluid produced the same symptoms as the choleraic discharges when injected into the venous system—acute gastro-intestinal catarrh, and in some of the rabbits death.

Similar researches with discharges disinfected by carbolic acid showed that the power of development in the elementary organisms was destroyed; the Cohn's fluid still remained clear at the end of twenty-four hours. On injecting the fluids into which the air had been conveyed through the disinfected excreta, the symptoms of poisoning with carbolic acid were produced; this is readily explained, when it is remembered that the current of air in the course of twenty-four hours must have carried with it a large amount of carbolic acid.

While these experiments were being carried on, the attendant, who had been exposed for some time to the emanations from the vessels containing the choleraic excreta, had a severe attack of gastro-intestinal catarrh, which recurred twice within a short period. His little daughter, who slept with him, had vomiting and diarrhea the day after he became ill. Five days after his illness, two cases of cholera (one of which ended in death) occurred in the house in which he resided, which had hitherto been free from the disease. During the microscopic examination of the choleraic discharges, Dr. Högzes had loss of appetite, a coated tongue, and a constant sensation of oppression in the epigastrium; after the researches were completed, these uncomfortable feelings disappeared.

The following are the conclusions at which Dr. Högzes has arrived:

1. Fresh choleraic discharges exert an injurious influence on animal organisms, as it seems in different degrees in different animals.

2. The principal or infallible evidence of injury after the introduction of choleraic discharges in any way, is more or less inflammation of the stomach and intestines.

3. An artificially induced gastro-intestinal catarrh renders animals more or less liable to be thus affected.

4. The same symptoms may be produced by the inspiration of a stream of air charged with particles from non disinfected choleraic discharges, as well as by immediate action on the stomach, intestines, or venous system; while the particles in choleraic discharges disinfected by carbolic acid, appear to be quite harmless.

5. A current of air, passing through non-disinfected choleraic excretions, carries with it cryptogamic elements, which vegetate abundantly in a favorable soil; while the same growths from discharges that have been disinfected by carbolic acid are incapable of multiplication.

6. Choleraic discharges freed from organized elements are capable, by reason of their chemic composition, of producing the same pathologic changes as they do when they contain the organized forms.[51]

The last conclusion established by the experiments of Dr. Andreas Högzes, is of importance in establishing the fact that the propagation of cholera does not depend on the organized elements. It may, however, at the same time be true that the fungi developed and propagated in the choleraic discharges, may become the vehicles or carriers of the poison of cholera. We have regarded the fungi found in the black vomit and other fluids of yellow fever, as in no respect the cause of the disease, but at the same time it may be regarded as probable that when developed in the poisonous matter of yellow fever they may become agents for its dissemination.

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[51] Centralblatt für die Malarinischen Wissinschaften, Nov. 1 and 8, 1873.

I have shown by a series of experiments[52] on the action of the poison of the American copperhead and of the American rattlesnake on living animals:

1. The primary and chief action of the poison of the American copperhead (*Trigonocephalus contortrix*) is upon the blood.

2. The poison of the copperhead is directly destructive to the colored corpuscles of the blood, altering their physical and chemical properties and relations, and rendering them unfit for the performance of their important offices in circulation, respiration and nutrition.

3. The poison of the copperhead appears to have an affinity more especially for the coloring matter of the colored blood corpuscles.

4. Under the action of the poison of the copperhead, the animal temperature is but slightly increased notwithstanding the profound changes inaugurated in the blood; and after the establishment and propagation of the pathologic changes the temperature descends.

5. The action of the heart is increased in frequency and diminished in force under the influence of the poison of the *Trigenocephalus centertrix.*

6. The profound alterations induced in the constitution of the blood by the poison of the American copperhead, give rise to passive hemorrhages into the cellular structures, and from the intestinal mu-

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[52] British Medical Journal, Dec. 13, 1873, p. 697–698. Medical and Surgical Memoirs, Vol. I, pp. 519–529.

cous membrane. This phenomenon recalls strongly the passive hemorrhages in certain febrile diseases, and especially yellow fever. Such experiments indicate that the black vomit of yellow fever is the result of the action of an irritant poison upon the blood, and gastric mucous membrane. We have also in these experiments an explanation of the mode in which dysentery might be induced by a poison introduced into the blood.

7. The poison of the rattlesnake (*Crotalus durissus*) acts as a local irritant upon the capillaries, and as a destructive agent in the blood and muscular structures, causing congestions and bloody effusions, and softening and disorganizing action of the muscular fibers.

8. The poison of the rattlesnake destroys by its direct effects upon the cerebro-spinal centers, and by its depressing effects upon the sympathetic ganglia, and muscular structures of the heart; and also by the changes which it induces in the composition of the blood.

If therefore a few drops of a secretion elaborated by special organs in living animals in a normal state be capable of producing profound disturbances in the blood and nerves and muscular systems; it can not be unphilosophical to refer the origin of certain diseases, as typhus fever and yellow fever, to the action of special poisons arising in the human body when subjected to certain conditions of food, climate, moisture and the foul air arising from overcrowding.

Another striking fact is that a deadly volatile poison may be generated during the fermentation or chemical and physical changes of certain vegetable constituents. Thus by a series of experiments[53] we have shown that the volatile poison hydrocyanic acid (prussic acid) induces profound alterations in the blood, and through this medium affects all the organs and tissues. We have endeavored to open the vast field of the action of well-known poisons, in order to elucidate the nature of the unknown febrile poisons.[54]

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[53] Medical and Surgical Memoirs, vol. I, pp. 279-330.

It is surely worthy of note that the most destructive vegetable product is the direct result of fermentation and is volatile.

Such facts may be plausibly adduced to show that a deleterious agent capable of producing yellow fever might be the product of animal and vegetable matters under different ranges of temperature. The conjecture has been hazarded that the poison of this disease resembles a volatile essential oil and which in conjunction with the albumin and ammonia of the atmosphere induces the phenomenon of yellow fever. They may also be adduced to sustain the hypothesis that the poison may be engendered in the human system under certain circumstances, and may be propagated through the medium of the atmosphere and by the agency of fungi and animalculæ.

THE END.

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[54] Et seq. Medical and Surgical Memoirs, vol. I, pp. 272-532.

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